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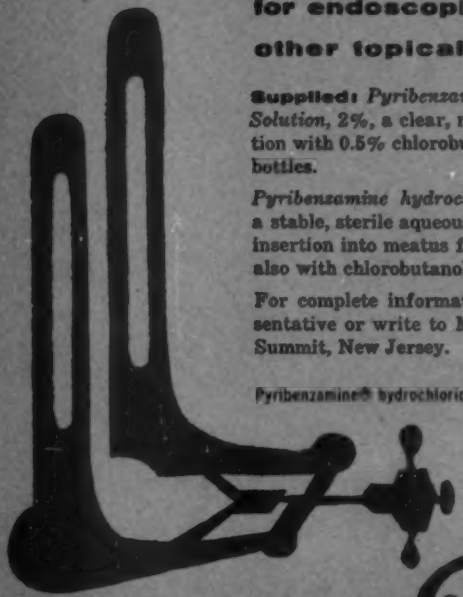

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THE
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VOL. LXV DECEMBER, 1955. No. 12

NUTRITION AND RELATED PROBLEMS.

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The physical ills that afflict mankind, it has seemed to us, fall into two groups. Of course, both of these are concerned with Nutrition—as is every phase of life, in sickness and in health.

Group I, the main subject of this editorial, includes the five subjects: Endocrine, Autonomic, Stress, Allergy—and Nutrition as it affects them or is affected by them. This group naturally concerns the physician, whether a specialist or a general practitioner, but lies particularly close to the daily practice of the otolaryngologist.

Group II consists of Infection, Neoplasm, Trauma, Heredity (including Congenital Abnormality)—and Nutrition as it concerns them.

"The writer must so write that his readers not only may, but must, understand." These were the words of Quintilian[1] nineteen hundred years ago. Let no reader suspect us of trying to prove anything; that is farthest from our thought or

interest. We are simply recording our own impressions from the study and care of the sick.

We were led to this concept of two groups by the following facts and experiences: To some, nutrition means simply food; if, however, it is defined as the sum of all processes whereby the body is nourished, Nutrition involves these other related subjects. In clinical practice, at any rate, Nutrition, Endocrine, Autonomic Influence, Stress, and Allergy are all closely interwoven. Nobody can study an article on any one of these five problems without being struck by its close relation to one or more of the others. The understanding of one is certainly not clear without an understanding of them all. Each of these five subjects is elaborately presented in the literature, *separately*, but it has seemed to us that they might be considered together, as the thumb and fingers of a hand.

Of all the problems in medicine, is there no other that belongs with these "five fingers"? The answer seems to be "No," for we feel that the others, of Group II, not intimately related to one another, may be considered as "on the other hand." To be sure, there are sometimes interrelations between the two groups; for example, trauma is definitely productive of stress. Consequently, the right hand and the left occasionally join in a hand-shake.

The approach to *treatment* of infection, neoplasm or trauma is on the average not difficult to determine; but we clinicians, absorbed in providing help for the patient, approach the five other problems with less assurance.

Now, to discuss this Group I. Of course, Stress, like Nutrition, is all-embracing—including the human soul! It is an active factor in the life of every man, no matter what disease he happens to have. Often, however, we think we are bearing a cross, when we are merely putting up with ourselves. Even Shakespeare challenges the physician: "Canst thou not minister to a mind diseas'd, pluck from the memory a rooted sorrow?"* Milton goes further in saying: "The mind is its own place, and in itself can make a heaven of hell, a hell of heaven."† Obviously, it is idle to limit Stress

* Macbeth, act V, sc. 3.
† Paradise Lost, bk 1, lines 254-5.

to a single group; yet it exerts its good influence (stimulus to achieve), or its dire influence *(dis*tress), each through the autonomic system, and so may conveniently be placed in Group I.

The *eosinophil* belongs to Group I, not to Group II.

It has been in our minds for a long time that, if only we could come to understand the *eosinophil*, we would have a better grasp, not only of nutrition but of the related subjects. "Comment is free, but a fact is sacred." At the moment we cannot know the facts about the eosinophil, and so various comments are in order.

The eosinophil and also, for that matter, allergy itself are attributed to the action of enzymes by Godlowski[2] in his book reporting his original work. It has been estimated that the protein of all the cells of the body is replaced every ninety days.[13] When the protein reaches the cell, if it has already been split into its terminal amino acids, all is well and it can be appropriated by the cell. If, however, it has not been sufficiently split into the innocuous amino acids, it changes the physico-chemistry within the cell. As he sees it, the tissue cell and also the leucocyte or histocyte can thereby be sensitized. For a substance to enter a cell, a particle of appropriate size must be adjacent to an open "cortical fenestra" and also a force must be available to drive it through the cortex into the interior of the cell. This is true of the original sensitizing invasion, and also of the shock dose which may come later.

This second assault can cause the liberation of toxins, with resultant edema and eosinophilia. To the rescue comes the adrenal, that "Watch-dog of the Treasury." In other words, toxic metabolites are released which evoke the pituitary-adrenal response. This reduces the exudate and cuts down the number of eosinophils. The factors in everyday living that produce stress can have the same effect. In passing, it is of interest that this process can result in harm as well as good, if it permits the spreading of a quiescent infection. A focus of infection, like tuberculosis, may be cut off from the rest of the body by a wall of tissue; hence corticosteroids can

destroy the barriers surrounding the focus. To Godlowski it appears that the eosinophil can be formed wherever the white cell or local histocyte can be insulted. Such a cell then bears the features of an insufficiency; it is no longer adequate for the performance of its normal function. In brief, he considers that this change into an eosinophil deprives the cell of its original morphology and primary function.

It would seem that some good thing might be said about eosinophils. In small numbers they are present in everyone, even those in apparent perfection of health; however, when the otolaryngologist finds them in the nasal smears and blood count of an allergy patient, he is inclined to liken the eosinophil to the title of a book well known to our parents and regard them as stained by the stigma of "The Scarlet Letter."* This, however, is by no means the orthodox view. The prevailing opinion is that we do not know where or how the eosinophil is born or what becomes of it when the adrenal cortex causes it to fade from the picture.

There appears to be a recognized sequence in Allergy, including all five of Group I: An irritant food or other offender sensitizes the cell, and by a later assault causes it to produce toxins with the allergic result—edema and eosinophils; the stress, through the autonomic system, causes the adrenal cortex to come to the rescue with its steroids poured into the blood stream.

Thus the eosinophil is an intimate factor in the interrelation of the five members of Group I—nutrition, endocrine, autonomic, stress and allergy. So once again these five seem like strands of different nature that are woven together into one single cloth.

In all other afflictions, like those from infection, trauma or neoplasm, the eosinophil is seldom in evidence. In prodigious numbers infection brings forth the life-saving leucocyte but not the eosinophil. Eosinophils are found in patients who have certain intestinal worms; but this is an infestation, not merely an infection. In the acute stage of trichinosis there are fever, high leucocyte and eosinophil counts and a strongly

* Nathaniel Hawthorne, 1804-64; "The Scarlet Letter," published 1850.

positive diazo reaction in the urine. Buylla, Llavona and Villaroya[3] encountered an outbreak and battled in with ACTH, giving the victims an average daily dose of 60 mg., for periods varying from 3 to 18 days. They saw quick relief from fever, dyspnea, muscular stiffness and nervous symptoms. There were relapses in some patients and a second course of ACTH was necessary. Only one patient was refractory to this therapy. It seems that the allergic inflammatory reaction of the tissues was modified or controlled by corticotropin. The use of the drug is stopped when the diazo reaction in the urine becomes negative.

As Macfarlane[4] expresses it: When a subject becomes sensitized to some allergen and is subsequently exposed to it, as in sufferers from asthma, hay fever, urticaria, or certain skin diseases, or in parasitic infestation with absorption of foreign protein, there is usually an eosinophilia, reaching in extreme cases as in trichinosis, as high as 30,000. Dent and Carrero[5] report similar counts in infestation by toxocara canis.

From breast feeding to old age the food problems of the allergic individual are presented and simplified by Bucher,[6] who feels that what seem to be insignificant changes in the diet will frequently bring about a desired result. Enzymes are complex compounds capable of speeding up chemical reactions, while the enzyme itself remains unchanged in the process. In brief, the enzyme is a catalyst. When faced with these problems of biochemistry most of us are only too pleased to have the facts simplified as far as possible. The pancreas, with the aid of other active glands and cells, normally secretes a sufficient concentration of enzymes to combine with all foods ingested, plus an excess which passes into the blood stream. Every normal cell in the body is thus protected against foods in the original state. A large number of active enzymes, distributed to all tissues and cells, is required for the processes of normal metabolism. The first group of enzymes digests the foods into their ultimate product—carbohydrates into sugar, fats into fatty acids and glycerin, and proteins into amino acids; however, these end-products of

protein digestion must be further acted upon before they become a part of living cells. A second group of enzymes reconstitutes the amino acids to proteins, comprising the protoplasm of living cells. Each enzyme exerts its action only upon substances whose molecules have a certain definite structural arrangement. The enzymes that act upon simple sugars are not capable of affecting complex sugars. There is no evidence that any single enzyme can produce more than one kind of ferment action. During the process of "hydrolysis," proteins are converted into proteose, peptones, polypeptides and finally amino acids. Hydrolysis is a process whereby a compound takes up oxygen and hydrogen in the proportion forming water, and is thereby resolved into another compound. The result is called splitting—protein splitting, fat splitting, starch splitting. On the market are several commercially prepared hydrolyzed proteins; these are less allergenic than the original product. They are less expensive than pure amino acids. They are obtainable with or without the addition of fats, carbohydrates or vitamins. Gelatin is the only protein that never produces an allergic reaction. When ingested, it very quickly breaks down to amino acids, so that the body is not dealing with the gelatin molecule but rather with the broken down products of gelatin which are not allergenic. Also when a protein is rendered less resistant to digestion, as by cooking, it becomes less allergenic.

Years ago, the pioneer, Rowe, stated that infection led the parade of all the ills, but that allergy was second.

All fields of clinical medicine are replete with disturbances of physiology that go on to conditions of pathology—sometimes irreversible. These step-by-step processes go through successive phases of cell-exhaustion or cell-starvation. Often the exhaustion is but a stage of starvation in that the period of replenishment never occurs, or is never allowed to occur.

Even though our knowledge is so limited, we know already that the complex human organism contains checks and balances in the form of enzymes and hormones to help restore the cell. Irregularities in autonomic innervation, in the orderly immunologic process and in smooth endocrine interaction, can

impede the restoration of these microscopic building blocks, the cells, just the same as the lack of nutrient substances.

Confucius would have cheerfully endorsed the later Chinese proverb, "Order is Heaven's first law."[7] In the composite whole those functions that *preserve order*—nutrient, endocrine, autonomic, environmental and immunologic—constitute the keystone of the *normal physiologic process*. The basis of their interaction at the cellular level is as yet poorly understood, but the importance of each is clinical fact. These functions within Group I are all *normal;* only when they fail in their normal duty is the patient sick. What a contrast to Group II! There is nothing normal in infection, neoplasm, trauma or hereditary taint. It is obvious that there is this pronounced difference between the two groups; one is a normal mechanism out of order, the other a direct affliction—a disease.

Thus it appears that Group I—malnutrition, endocrine imbalance, autonomic dysfunction, stress and allergy — are precursors of disease processes, so interwoven and similar as sometimes to defy separation. It is probable that their malfunction sums up as cell starvation, irreversible change and established pathology.

As with most surgical specialties otolaryngology grew up on the surgical removal of diseased tissue. As our specialty comes of age and justifies its existence, its prime focus has happily shifted to the recognition of disturbed function and the prevention of disease.

As a source, the otolaryngologic literature is poor in the fundamentals. Most of the basic work is done in other medical fields. Hence the aim and, perhaps, the justification for an annual report on Nutrition — this the fourteenth — is the presentation of our concept of the Two Groups in Medicine, and of the changes and additions recently wrought in it. At last Nutrition begins to interest all physicians. It is our hope that, to the already accumulated wealth of gold, we may be contributing a bright penny.

In presenting the following, we recall the words of Montaigne[8]: "I have gathered a posy of other men's flowers, and have brought nothing of my own but the thread that ties them together."

NUTRITION.

Escorting us back to the remotest ages of the past, before the appearance of the first cell, Szent Györgi[9] says, paradoxically, that the longest period of life was the pre-cellular era when there was no life. From the first cell Nature built higher and more complex organisms, not making the cell more complex but multiplying it and allowing the new cells to differentiate. Every cell is the center of its own Universe, which is divisible into two parts—the inside and the outside, with a plane separating them, called the "membrane." Being permeable, this membrane permits the passing of ions to and fro; and its permeability, influenced by certain steroids, regulates the intracellular atmosphere which controls the cell's internal machinery. The passage of ions, in and out through the membrane, is a vital process. The potassium ions are dominant. To the question, "Is the potassium ion free or not free?" Szent Györgi replies, "Free? Yes," and "Not free? Yes." He compares it to a married man, "allowed to do anything his wife wants him to do." So many potassium ions passing in, so many passing out; there is no static equilibrium, but the situation constantly changes, bringing to mind Karr's century-old epigram, "Plus ça change, plus c'est la même chose."* There is ample evidence that, if a heart dies, poisoned by the products of infection, the only thing that has gone wrong is the membrane of its cells which has become too permeable to potassium.

One of our closest friends was Manager of the Research Study Club since its beginning in 1922. In him we have a picture of disturbances of all five members of Group I, Nutrition, Endocrine, Autonomic, Stress and Allergy (though little of the last). He was well known to a large number of the readers of this magazine. Those who have attended the

* "The more it changes, the more it remains the same." Alphonse Karr, 1808-1890.

Midwinter Convention will not forget this genial man with the monstrous abdomen. Each one wondered what had caused his trouble. Through the years the only explanation we have found is "war stress." In 1917, when he enlisted in the service at age twenty-three, he was well built, an expert swimmer and a speedy trackman. He spent eighteen and one-half months overseas with the Rainbow Division, much of the time under fire, enduring much mental and physical strain. His was a very sensitive soul. For a year or more after the war it was extremely difficult for him to meet people—he was intensely nervous, cried frequently and suffered violent headaches, some so severe that "he was out of his head." "Shell shock" was the term then in use. At this time he began to gain weight, pound after pound, until he weighed 360, his obesity being chiefly in the abdomen. It was not from overeating; in fact he tried dieting, exercise—everything—without success. All his physicians said "the trouble is glandular," but no treatment was of avail except insulin for the diabetes which had developed. At age fifty-eight he suffered an acute myocardial infarction and died nine days later. The autopsy disclosed severe generalized arteriosclerosis, coronary thrombosis with massive myocardial infarction, fibrosis of the islands of Langerhans and obesity; unfortunately, it did not include an examination of the brain, therefore there was no study of the hypothalamus.

Obesity, as a problem, should interest all physicians. It even concerns some of them personally. If the average weight at ages 25 to 30 years continues to be maintained, it confers a maximal life expectancy. For his height and build, no one throughout life should weigh more than the average person weighs at age 30. Frequently the physician fails to emphasize the seriousness of obesity, so in presenting this state of affairs Rynearson and Gastineau[10] urge us to develop a missionary spirit and zeal in its correction. The facts are clear. In war time. countries short of food have noted a decrease in degenerative diseases; in this sense they are blessed in being without their usual food supply. These authors contend that. as fat comes only from food, obesity results simply from eating more than is required. Too much food creates a stress

with which the body has only limited ability to cope, so when the body is carrying a load greater than it was designed for, it is like a passenger car motor being used to propel a truck. Damage to the hypothalamus also has a striking effect in producing obesity; this suggests that an important center for appetite regulation resides there. The effect of odor and sight of food in arousing appetite, and even of mere memories of good meals, is familiar to everyone. After damage to the hypothalamus, obesity results from the voracious, ravenous appetite which begins abruptly after the injury. Such injury has been shown to interfere also with other important self-regulating functions—temperature control, water balance, gastro-intestinal function and even sleep. Since some lesions of the hypothalamus will produce obesity and others will cause genital hypoplasia, it appears probable that the responsible centers are separated anatomically. During periods of stress some people smoke, some drink, while others eat and become fat. Supplemental thyroid has been used in obesity since 1894. It gained early favor, until time proved that hypothyroidism was only occasionally associated with obesity. As to exercise, one may lose one pound by walking thirty-six miles; however, exercise tends to increase the appetite. In excess of basal requirements, hard physical labor may cause additional consumption of as much as 2,000 calories a day.

In contrast to the above, there are many who consider that this frustrating problem has too often been dismissed as a simple habit of overeating, presumably to be cured by discipline. It is believed by Van Itallie, Mayer and Stare[11] that obesity can best be placed in proper perspective when viewed as an end-product for which many different factors can be responsible. To be sure, obesity can develop only if there is a prolonged imbalance between energy intake and output. To understand the cause of such an imbalance, the normal mechanism of food intake must be understood, and our present knowledge concerning this important mechanism is sketchy.

In Britain obesity is unquestionably the commonest nutritional disorder, and Meiklejohn[12] considers it responsible for more ill-health than all the vitamin deficiencies put together.

The American male, if twenty-five pounds overweight at age 45, has his life expectancy reduced by 25 per cent. It would seem that for those who "laugh and grow fat," the laughter is frequently hollow. Endocrinology has given the physician a useful escape from the distasteful task of telling his patients they are greedy; it can all be "blamed on the glands." The lack of any positive evidence has led to a return to the earlier belief that the vast majority simply eat too much, either because of sheer gluttony or because of a disorder of the hypothalamus; yet some who eat surprisingly little still become fat. There is undoubtedly an endogenous or metabolic cause of obesity, as well as the exogenous one of simple overeating. The only possible origin of the fat is food; it does not come out of the air. The unknown factor is the metabolic process in storing it. Perhaps normal people have some means, as yet undefined, of disposing of surplus calories. This may be impaired in the obese. Certainly many normal people regularly take more calories than their bodies need, yet without becoming obese. In brief, what the normal body would eliminate as heat, the obese body stores as fat. The hypothalamus is undoubtedly concerned with the regulation of appetite; damage to it can directly produce an excessive appetite. Finally, it also seems that overeating can be due to stimuli from the cerebral cortex, directed to the hypothalamus which, in turn, controls the numerous neural and endocrine mechanisms affecting appetite and the utilization of food. The complicated relationships between the mind, the hypothalamus, the anterior pituitary, the pancreas and no doubt other regulatory mechanisms are imperfectly understood; but we already have sufficient insight to regard many cases of obesity as suffering from a true psychosomatic disease.

In their study of older people Shea, Jones and Stare[13] find that women are able to maintain their weight on a daily caloric intake of 1500 calories or even less. For men it is nearer 2000. Further they suggest that the caloric intake be reduced 5 to 7 per cent for each decade beyond age 25. Thus a moderately active man who was maintaining his weight at age 25 with a daily consumption of 3000 calories should limit himself to 2400 at age 65 and to 2300 at age 75.

If he is no longer active, as in most instances, the caloric allowance should be decreased even further. Protein is in a constant state of flux in the aged individual just as it is in the young. It has been calculated that there is a complete turnover of body proteins every ninety days as the body tissues are broken down and repaired. Since excess protein is *not stored* in the body, regular daily consumption is necessary. With age, the proportion of total calories derived from fat should be decreased. Fat is more slowly digested than carbohydrates and proteins, but there should be a sufficient amount of the essential fatty acids as these may play a role in maintaining healthy skin. Constipation is no more common in the aged than in the young. Old people just talk about it more.

Under the broad heading, "Nutrition and Disease," Spies[14] has assembled reports of some ninety cases. One of these illustrates the wisdom of treating the patient for general welfare, and not concentrating on just one aspect. The patient suffered from combined hypertension and obesity. Her weight was 215 pounds and her blood pressure 178/110. On a reduction diet, in five months she lost 21 pounds and in the next three months another 15 pounds; concurrently there was a steady drop in blood pressure, to 108/72. Her basic trouble was clearly obesity. It is said that 20 per cent of our population are obese.

The separation of the process of senescence, *per se*, from the chronic diseases seems to Davidson[15] to be difficult; but he regards it as common knowledge that many of these degenerative diseases are nutritional and can be so treated. As one would expect, there is an increase in fat content of the body with aging, particularly in women; this is associated with a decline in muscle mass. With increasing obesity there is a marked increase in mortality, due largely to cardiovascular and renal disease and to diabetes. There is also some evidence that weight reduction is accompanied by a reduction in mortality. It is recognized that psychic stress may lead to overweight, by operating through the hypothalamus, just as injury to certain areas of the hypothalamus in animals will cause an increase of appetite and weight.

It now seems that intestinal bacteria definitely contribute to the wellbeing of the host by synthesis of water-soluble vitamins. This has been well established in ruminants and Stokstad[16] shows abundant data suggesting a similar synthesis of vitamins in the intestinal tract of single-stomached animals. The recognition that antibiotics can increase the growth rate of animals has given a new insight into the function of intestinal flora and has naturally given rise to much speculation. Certain antibiotics produce a decrease in some bacteria and an increase in others. The marked effect of antibiotics on slow-growing pigs naturally suggested a similar experiment for slow-growing children, of course with careful controls. Antibiotics were given to premature infants, and also to poorly developed and undernourished children. With twins and triplets, the antibiotic was given to the weaker or weakest one. The results on animals and children alike have been sufficiently good to warrant further tests.

It is logical to expect that the rate of growth of an animal will be increased when infection is eliminated. This, in a nutshell, is the reason given by Jukes and Williams[17] for feeding antibiotics. They found that adding small quantities of antibiotics to the diets of young animals not only served to control certain obvious infections but also produced unexpected increases in the growth rate even when the animals showed no evidence of infection. It is suggested that microorganisms which slow down the rate of growth must occur commonly in the digestive tracts of animals. Just a trace of antibiotics has created a near-revolution in poultry and live stock raising. The animals require less food and yet show an increase in size and weight. It seemed from the first that the effect of the antibiotics on growth was due to their attack on the intestinal flora. This growth effect is produced by several substances but the only known property which they have in common is their antibacterial potency. The growth of human beings takes place so much more slowly that such growth responses to antibiotics are not to be anticipated in infants. A pig grows to a weight of 50 kg. in about one-fiftieth of the time taken by human beings to attain this weight. The effect of antibiotics is not obtained in environ-

ments in which bacteria are absent, such as in the chick embryo or in "germ-free" chicks, and the growth effect may be reduced when the animals are kept under highly sanitary conditions. It seems evident, therefore, that certain antibiotic-sensitive intestinal microorganisms interfere with the utilization of food. The tissues of antibiotic-fed animals are of normal composition. The practice of feeding antibiotics to farm animals is now widespread. No untoward effects on public health have resulted. Detectable amounts of antibiotics are not found in meat even when the animals are fed antibiotics at levels far higher than usual; furthermore, many antibiotics are destroyed by cooking. The daily administration to children of small amounts of aureomycin for prolonged periods was found to have no unfavorable effect.

To honor Albert Szent-Györgi, Nobel Prize winner, the New York Academy of Sciences conducted a conference on "Bioflavonoids and the Capillary." His investigations of the properties of extracts of citrus fruits, especially the pulp and peel of the lemon, began some twenty years ago; he determined the presence of a substance which so acts upon the capillary wall as to lessen its permeability, and believed it to be a single entity which he named "Vitamin P" (for permeability). Later work dispelled the concept of a true vitamin and revealed the existence of substances characterized by a yellow pigment and called flavonoids (Latin *flavus*-yellow) or bioflavonoids; in them lies the power to reduce the permeability of capillary walls and thus to control or lessen capillary bleeding. The conference brought together workers in the citrus and pharmaceutical industries, agricultural and university laboratories, and clinics . . . Zweifach[18] enumerates the five parts of the capillary wall; the one through which pass large molecular aggregates and formed elements is the intercellular cement . . . In patients under coumarin anticoagulant therapy, Brambel[18] found evidence of capillary fragility, such as ecchymotic areas, in five percent; by the use of hesperidin and "C" in combination (the now generally accepted practice) bleeding was arrested and there was rapid clearing of the ecchymoses . . . The ravages of rheumatic fever were depicted by Rinehart[18]; the initial re-

action to the "rheumatic injury" involves a swelling of the connective tissue ground substance, to whose economy "C" and the bioflavonoids are important; epistaxis, a commonly encountered complication, is reported to be reduced in its incidence and severity by these agents; the records of the Royal Navy support the belief that a deficiency of "C", in the presence of hemolytic streptococcal infection, disposes to the development of rheumatic fever; admitting that direct evidence is limited, Rinehart finds much to support the view that the use of "C" and bioflavonoids, in prophylaxis and treatment of rheumatic fever, is basically sound . . . Likewise Javert[15] has found, in cases of repeated abortion, that the lack of "C" and hesperidin is often a determining factor and that it can be overcome by their use . . . Poliomyelitis, in turn, offers occasion for the same mode of treatment; Boines[16] adopts the dictum "an intact capillary system means a solvent body," and reports that in all his cases of severe acute infection there was capillary fragility and that "C" with hesperidin effected improvement in 80 percent, in an average period of five weeks . . . It remained for Youmans[18] to summarize the clinical aspects of bioflavonoids and ascorbic acid; a deficiency of "C" can be blamed for bleeding from the capillaries; its presence is necessary for the integrity of their walls; the role of the bioflavonoids is inferred from indirect evidence only; we need additional indices, additional measures of determination, in order to arrive at a more clear-cut understanding of their action and mechanism. However, he concludes, when one is dealing with human subjects and human illness, one is forced into empiricism more than to one's liking and must depend primarily on the therapeutic result . . . Fittingly Szent-Györgi[18] was the last speaker; he deprecated the hard and fast division of the substances under discussion into "vitamins" and "drugs", for perhaps there is something between. Are the yellow pigment-bodies normal constituents of the cell? He knows of no proof, and calls for an attitude of caution. He has extracted from the thymus a strongly yellow substance, giving most reactions typical of flavonoids; further search may lead to a better grasp of the thymus' function. "Flavonoids represent one of the most exciting,

broad and hopeful fields of biological inquiry, and I am glad to close on such an optimistic note."

ENDOCRINE.

At the turn of the century a student was asked if he knew the cause of diabetes. "I did know once, sir, but I have forgotten." The Professor said "What a pity — now no one knows." To this day no one knows completely, but Bishop[19] says that through endocrine studies the secrets of diabetes are gradually being revealed. Blood glucose remains remarkably constant; it is disposed of by being converted into fat and muscle glycogen. This is facilitated by insulin—indeed this is probably the principal function of insulin. Sugar is stored in the liver as glycogen. The conversion of liver glycogen to sugar in the circulation is largely adjusted by the blood glucose level. When it tends to fall more glycogen is released; if it tends to rise more glycogen is stored. When the blood glucose is converted into tissue glycogen, the glucose combines with phosphoric acid. The most important role is played by the enzyme, hexokinase, and its action is helped by insulin. In normal circumstances muscle glycogen is the main source of body energy; the glycogen is oxidized, yielding carbon dioxide and water. Should there be limited glycogen stores, fat is mobilized and acts as the main source of energy. Probably the most important effect of insulin concerns the utilization of blood sugar by the tissues. The formation of liver glycogen is greater than that of tissue glycogen.

So we do not know how insulin works. In fact, to those who have a small acquaintance with the vast subject of diabetes it is almost a shock to have Stadie[20] state that in the light of the evidence presented it is impossible at present to espouse one theory of insulin action to the complete exclusion of any other; yet it is with reluctance that one would abandon the hope that the interactions of insulin will become explained upon the basis of one single mechanism.

An adenoidectomy—will the child bleed profusely in the hours following? An epistaxis—what will happen after the

packing has been removed? Menger[21] has used conjugated estrogen substances in such crises when routine procedures had failed to arrest bleeding. He reports six cases of epistaxis. In one, the bleeding had persisted for three days and transfusion, "K" and other agents did not stop it but 20 mg. of conjugated equine estrogens given in the vein succeeded in doing so in one hour; a minor bleeding three days later yielded to the same treatment in 30 minutes. He reports also ten cases of bleeding in young children, following the removal of an adenoid; the estrogen was given in the gluteal muscles, in amounts of 5 or 10 mg. according to the weight of the child; no bleeding after a period of from one-half to two hours. It may be that the female hormone restores the integrity of the blood vessels; it may speed up the clotting time or provide some missing factor; it may stimulate the release of thrombocytes into the circulation. Menger admits the empiricism of the therapy, but bases his estimate of its value on his own experiences.

In meningococcic infections two factors dominate the course, the infection itself and the action of the adrenal cortex. Bartolozzi and Borgheresi[22] find that in the fulminant cases the protection afforded by corticotropin is seriously impaired and there is a correspondingly high eosinophil count; by the use of cortisone the patient is enabled to derive benefit from antibiotic therapy. The effect of the cortisone is reflected in a falling of the eosinophil count, and a regression of the state of collapse; in this period the victim is actually kept alive until the antibiotic can control the infection. In more moderate infections the eosinophil count is low, indicating good adrenal function; cortisone speeds the recovery in some cases, not in all. Accordingly the authors ascribe high value to the use of cortisone in severe fulminant infections, less value when the stress of the disease is mild. The eosinophil count reflects the activity or non-activity of the adrenal cortex and is a positive indication for the use of the hormone when its non-function threatens failure to meet the demands of the infection.

AUTONOMIC.

In the light of recent knowledge it now seems natural to

think of the hypothalamus and the pituitary as almost inseparable parts of a functional unit. The main structural features of the pituitary are obvious even to the naked eye, but Kennedy[23] notes that the structure of the hypothalamus remains something of a mystery. Early experiments, of which there were many, were inconclusive because the hypothalamus was always approached from below, so that the pituitary also was inevitably damaged. The crucial experiments demonstrating that the hypothalamus could be damaged so as to cause obesity, introduced an electrode from above, without any harm to the pituitary, so now there is no reasonable doubt that the cause of the obesity often associated with damage to structures in the pituitary region lies in the hypothalamus and not in the gland itself. The most important factor is an increase in food intake; and bilateral lesions of the ventromedial nuclei of the hypothalamus were necessary to produce this result. The original work was done on the rat and confirmatory studies have been done on the cat, the dog and the monkey and it is a reasonable assumption from clinical evidence that a similar mechanism operates in man. At any stage after the development of the obesity, the rat can be reduced to the same weight as its control by starvation; if it then receives excess food again, the whole picture is reproduced and the animal again reaches about the same degree of obesity. Determination of the fat content of the most obese rats shows that the fraction of their body formed by fat is almost identical with that in very fat women as well as in fat pigs. Initially the feeding is voracious, but the animal's behavior becomes more normal as it becomes fatter.

In higher animals the sensitive mechanism controlling food intake is thought to be located in the brain, since all of the reactions for finding and eating food are directed by the central nervous system. If there is only one sensitive region having this function, Brobeck[24] considers that it is probably located in the hypothalamus. Within this remarkable hypothalamus, two mechanisms appear to be separable; one, in the lateral regions, is required for eating and has been called a feeding center; the other, in the medial hypothalamus, evidently acts to inhibit either these lateral mechanisms, or the

lower mechanisms which take part in feeding. The lateral mechanism may be responsible for appetite and the medial mechanism for satiety.

STRESS.

"Galen's art heals only the body,
But Maimuni's skill heals both the body and the soul.
. . . . . .
For evil and ignorance flee from his presence;
When Maimuni arrives, all suffering departs."*

Compressed into five pages of a popular magazine, Ratcliff's[25] story of Hans Selye recounts his development of the concept of Stress as a prime cause of disease. Even in his student days at the German University of Prague, Selye rejected the teaching that most disease is caused by bacteria and nothing else counted very much. Hormones and stress opened unknown territory. He came to this country and for a time studied at Baltimore. Later, he moved to Montreal where he engaged in research for ten years at McGill University; in 1945 he became head of the Institute of Experimental Medicine at the University of Montreal. Selye exposed animals to stress in one form or another and saw them sicken and die; he traced the effects of the stress through pituitary and adrenal glands to arterial and renal disease, to arthritis and to damaged lymphatic systems. The secret lay in endocrine imbalance, a story now familiar to every medical reader but ever new in its significance.

In his book on Stress, Wolff[26] views the many problems involved as they affect individuals and peoples. Wartime conditions in Norway in the late war resulted in a striking rise in the incidence of hyperthyroidism, especially in the female part of the population. In Central India society is undergoing a rapid cultural change among the more opulent citizenry, whereas the villagers continue to follow the standards of their forebears; the "Westernized" Indians of the cities are more subject to intestinal disorders, to neurocirculatory asthenia

* From a tribute to Maimonides, written by Alsaid Ibn Sina Almulk when he learned of Maimonides' death in 1204.

and to asthma than their ignorant and underfed fellow-countrymen of the small communities and farms.

The note of group psychology is also sounded by Wolff[27] in a "Lecture to the Laity." Man is a tribal or group creature, with a long period of dependence and development. He is dependent for his very existence upon the aid, support and encouragement of other men. He lives his life so much in contact with men and in such concern about their expectations of him that he is jeopardized as well as supported by his fellows; indeed, he may feel more threatened by cultural and individual pressures than by other environmental forces. He must be part of the tribe, and yet he is driven to fulfill his own proclivities. These pressures and the conflicts they engender are ubiquitous and create a large portion of man's stress.

Stress from terror reaches the ultimate in its effect on the ignorant and superstitious. Hume[28] tells of a tragic outcome in the case of a husky African native who underwent a simple hernia operation at the hands of a missionary doctor working among the Zulus. On the third day after the operation he saw passing his hut a man with whom he had had a quarrel; he was convinced that the man would "invoke strong medicine" to work him ill. No assurance of the surgeon could influence him. "I'm going to die," he kept repeating—and die he did. He passed out as a result of starvation and violent apprehension —scared to death.

More and more our profession is invited and urged to consider the bearing of mental stress on bodily health. The problem, as it presents itself to a clergyman "of a somewhat scientific turn of mind, although not a scientist," is the subject of an address delivered before the American Medical Association by Peale[29] at the installation of its president. Physicians recognize that a long held mass of unhappy thoughts can produce physical symptoms of illness. Man is soul as well as body; in the treatment of physical symptoms full effectiveness becomes more likely when the emotional or spiritual need is cared for. In a cited instance the patient resented his physician's searching questions — "his

morals had no relation to his physical condition." Wrongdoing was finally brought to light and its rectification led to great improvement in the man's physical state. In general, caution is still indicated in the acceptance of non-medical healing, but all realize that amazing phenomena do occur which are outside the framework of man's understanding.

It would be interesting to know what reaction occurred in the minds of all those doctors. The patient asks, "What does it mean—mind over matter?" Here we are on solid ground. The popular word came to be "psychosomatic"; when first used, many patients thought it signified a new approach, was, in fact, a real discovery. Of course, the only new thing was this word, now applied to the old, well known relationship. As physicians, we have always known that the mind is Captain of the Ship; the only new thing is that we are becoming better acquainted with the Executive Officer, the Hypothalamus. It is a surprise to the patient when we tell him that we have had 5,000 years of psychosomatic medicine. Finally came the amazing medicine and surgery of the *body*, which, especially in the last fifty years, has resulted in longer life, a healthier race and a far better habitation for the soul of man.

Current use of the word Stress has to do largely with such factors as anxiety or grief, which are not connected with laboratory experimentation. Dubos,[30] however, seeks to induce stress in mice by giving thyroxin or other chemical agents, or by withholding food for 36 to 48 hours. The most effective way of lowering resistance to infection is to starve the animal for 48 hours, before inoculation with bacteria. He put one group of mice on a standard Sherman diet, a second on a diet low in protein and then on a starvation period of 48 hours. He injected bacteria and allowed ten hours to elapse, with the following results: The well-fed group showed sterile blood and a few organisms in the spleen and the liver; the starved mice had bacteria in the blood and in very high counts in the same organs.

ALLERGY.

The evidence that histamine plays a part in anaphylaxis

is so strong that there is hardly anyone today who would have the temerity to deny it; but Gladstone[31] suggests that it is not the only factor concerned. When histamine is injected into an animal the resulting condition of shock does present certain differences from anaphylactic shock. One difference is the lessened blood coagulability due to the liberation of heparin from the liver, which is absent in histamine shock. Shock produced by peptone is more like anaphylactic shock than that produced by histamine. The main manifestations of shock can be accounted for by contraction of smooth muscle and damage to capillary endothelium. Anaphylaxis, therefore, is a highly artificial condition brought about by the injection of a large amount of antigen directly into the circulation of an animal that has been sensitized. There are certain natural diseases which differ from anaphylaxis in a number of ways but in which there is definite evidence of a similar underlying mechanism. It is these diseases, mainly studied in man, that are termed "allergic."

In 1845 Wharton Jones described certain white blood cells of distinctive appearance, with double refractory granules. Thirty-four years later Ehrlich described their identification with acid stains. Since Ehrlich's day Samter[32] has estimated that close to 10,000 papers on this subject have been written; yet our knowledge of these cells, the eosinophils, is still deficient. When their number in the circulating blood is decreased by ACTH or the hormones of the adrenal cortex, what becomes of those lost from the circulation? Why is the reduction in the count so consistently in the neighborhood of fifty per cent? We do not know. Samter has demonstrated that lung tissue from sensitized guinea pigs can be transferred to normal pigs and effect an eosinophilia; he expresses confidence that an "eosinophilic factor" exists, though not yet discovered.

The effectiveness of ACTH and cortisone in the treatment of asthma led Kuhne, Schmidt and Kania[33] to employ cortisone alone or combined with fever therapy in a series of forty cases. Artificial fever was brought on by a turpentine abscess in thirteen, by injection of milk or a mixture of non-pathogenic

bacteria in normal saline solution in fourteen; in the other thirteen cases, cortisone was given alone. The combination of turpentine abscess fever and cortisone gave especially gratifying results; this was the more striking as the patients in this group were suffering from severe continued asthmatic spasm. Three cases were failures, but in general the tendency toward attacks was reduced. The presence of eosinophilia proves lowered function of the adrenal cortex and calls for replacement therapy with cortisone.

MINERALS.

For the localization of brain tumors, Amyes, Deeb, Vogel and Adams[34] employ a radioactive isotope, iodine or phosphorus, and two kinds of Geiger counter. The standard type of instrument is used in the diagnostic survey, and held in close proximity to the skull; the smaller, more sensitive scintillation counter is introduced into brain substance through a surgical opening, on some occasions a small burr hole. This latter instrument is a needle Geiger tube, 2 mm. in diameter, with a sensitive tip; it makes possible a localizing of neoplastic tissue with a minimum of damage to the brain. The external approach has its limitations, being of value for tumors in the cerebral hemispheres but not for those in the posterior fossa or the midline. The scintillation counter is brought into use in the first stages of surgery; in many cases it reveals the exact site and dimensions of the tumor. Of fifteen cases of expanding intracranial lesions, its telltale click gave the needed information in fourteen; in the fifteenth case the lesion was an abscess.

A long-range study of the value of fluorides in the prevention of dental caries was begun seven years ago in the Passaic General Hospital. Expectant mothers receive a fluoride during pregnancy; the child takes it later, until calcification of the permanent teeth is complete. At the time of the birth, samples of blood are taken from the umbilical cord and the placenta; the fluoride content is calculated and checked against similar samples from "control" patients, who have received no fluoride except what happened to be in their drinking water. The "cord blood" shows in the average pa-

tient a much higher fluoride content than is found in the "cord blood" of the controls, by as much as 175 to 250 per cent. The placental blood likewise shows an increase, but in less degree. Feltman and Kosel[35] present an interim report. The study is in progress, and the final results will not be determined until the investigators can evaluate the resistance of the permanent teeth to decay.

The role played by minerals in the human economy, a matter of interest to everyone, receives due attention from Davidson.[15] The past decade has brought into great prominence the question: What part has Fluorine in the prevention of dental caries? The fluoride content of foods must not be overlooked as a source of supply of this element; for example, certain baby foods with added bone meal may contribute a considerable amount. The answer is still to be determined. In the matter of Iron, it is established that iron is bound by a specific globulin, beta-l. Once iron stores are depleted they apparently are rebuilt slowly, whether the source is food or medication. A baby has adequate iron supplies for its first six months; but, if it is maintained on milk alone, it may develop an iron deficiency anemia. Copper, in turn, is essential for the synthesis of hemoglobin, but our diets usually contain enough copper for our need. Cobalt is an essential part of the "B12" molecule. Lack of cobalt in sheep creates a deficiency preventing synthesis of "B12" and leads to severe anemia and weight loss. Responses to cobalt and to "B12" seem to be similar, if not identical. From animal experimentation Zinc is deemed a necessity for normal growth. It appears to be related to insulin storage and activity.

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CARCINOMA OF THE LARYNX, CLASSIFICATION AND RESULTS OF TREATMENT.*†

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With the advent of the new radiotherapy machines and their increasing use in clinical work, it has been considered advantageous at this stage to evaluate the results of treatment by the older methods. Although so far there has been no definite evidence that the cure in relatively superficial lesions, such as cancer of the larynx, is likely to be any greater with the newer types of radiotherapy[1], obviously some standard must be set in order that their true value may be estimated. The results obtained by treatment with former methods of radiation as well as by surgical procedures are presented. Such results will form a basis for comparison with newer methods.

CLASSIFICATION.

To the present day there is considerable confusion as to the classification of tumors of the larynx. It is necessary, therefore, before going further to discuss this problem.

Probably the most widely used system is one devised in the nineteenth century: Krishaber[3] and Isambert[2] were responsible for the division of laryngeal tumors into *1.* Extrinsic; *2.* Intrinsic; and *3.* Subglottic groups. There is some difference of opinion as to which of these gentlemen was responsible for the introduction of the extrinsic group.

The curious feature, however, is that this system has survived so long, especially as it was conceived within two decades of the inception of the laryngeal mirror.

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In order to discover just what the terms in this classification mean, the literature of various authorities has been reviewed.

The *Subglottic* group presents little difficulty. It is agreed that tumors in this group arise between the lower border of the cricoid and the under surface of the true vocal cord. They are notoriously "silent" for a long period and the prognosis usually is poor.

The *Intrinsic* group. There is a great divergence of opinion as to the extent of this group. The lower limit is generally accepted to be the free margin of the true cord; however, the upper limit varies considerably. Some take the true cord as the limit and use "intrinsic" and "cordal" as almost synonymous (Walsh,[4] Friedberg and Vollner), others take the free margin of the false cord (Hayes Martin)[5], and some accept the upper surface of the vestibular fold (Negus)[6].

In the classification where "intrinsic" is applied to cordal lesions, "endolaryngeal" is applied to lesions involving the ventricle, ventricular bands, arytenoids, interarytenoid fold or crossing the anterior commissure.

Due to the difference in symptoms, degree of differentiation of tumor, rate of progress of lesion, and prognosis, it seems advisable to separate the cordal lesions from others arising within the lumen of the larynx.

The *Extrinsic* group. This term has been used to describe tumors invading the inner surface of the larynx above the very indefinite extent of the intrinsic group. In Walsh's classification it is applied to lesions arising superiorly to those in the "endolaryngeal" group. It is also applied to lesions on the outer aspect of the larynx, such as the pyriform sinus and postcricoid region.

On consideration of the physiology, histology, symptomatology and results of treatment it would seem essential to regard lesions of the upper aperture and outer surface of the larynx as pharyngeal rather than laryngeal. In recent years

this has been the view of Baclesse,[7] Cach, Hautant, Leborgne,[8] Lederman,[9] Lerou, Robert and Wilson.

With this considerable confusion as to classification the anatomical sites of carcinoma of the larynx as described by the International Committee for Stage-grouping in Cancer and for the Presentation of the Results of Treatment of Cancer (referred to as I.C.P.R.) in Copenhagen in July, 1953, is extensively welcome. The report of the committee has not yet been officially accepted, but is a guide to the modern outlook on the problem by the radiotherapist. Such a classification (Table I) also offers advantages to the surgeon.

TABLE I.

ANATOMICAL SITES OF CARCINOMA OF THE LARYNX I.C.P.R.

Carcinoma of the Supraglottis.

Carcinoma of the Glottis (Vocal Cords).

Carcinoma of the Subglottis.

Carcinoma Occurring in Borderline Areas.

The lesions in this fourth group "arise at or near the upper border of the larynx in which on examination it is impossible to state accurately whether the original lesion began within or without the larynx. Such lesions should be included with the pharyngeal carcinoma."

The group "Carcinoma Occurring in Borderline Areas" is still rather vague. Lederman[9] giving sound reasons had previously suggested that the epiglottis be divided into infrahyoid and suprahyoid portions; growths involving the suprahyoid portion should be considered epilaryngeal tumors of the pharynx. Thus the supraglottic area includes the ventricle, ventricular fold and infrahyoid portion of the epiglottis. This puts a more definite limit to the supraglottic area and should reduce the number of cases being placed in that group.

STAGING.

The staging as well as classification has been of various types but the I.C.P.R. method, previously described by Leborgne,[8] is the one used in this series and is shown in Table II.

TABLE II.

CLINICAL STAGES IN CARCINOMA OF THE SUPRAGLOTTIS GLOTTIS AND SUBGLOTTIS (I.S.P.R.)

Stage I. Carcinoma limited to the mucous membrane. Full mobility of the larynx retained. No nodes palpable.

Stage II. Carcinoma, infiltrating but not extending beyond larynx. Mobility of larynx impaired or lost. No nodes palpable.

Stage III. Carcinoma extending beyond larynx, or nodes palpable but movable.

Stage IV. Involvement of skin, or nodes fixed, or distant metastases present.

The term "mobility of the larynx" should be noted. The obvious meaning, mobility of the whole larynx on deglutition or palpation is certainly not intended nor does it mean movement of the vocal cords in all sites. In the supraglottic lesions it refers to the movement of the ventricular fold; in the glottic lesions, to movement of the vocal cord; and in the subglottic lesions it must refer to cord movement. This latter is a weakness in the system but as this group is relatively small it is not very important. Certainly this system of staging is an improvement on that previously accepted, the system of Nielsen[10] where the glottic lesions are adequately dealt with but the subglottic are arbitrarily allocated to Stage III and supraglottic lesions to Stage IV. Thus in the Nielsen staging, results of treatment in Stages I and II give higher results because the unfavorable noncordal cases are excluded and the survival rates in Stages III and IV are raised by including the early non-cordal lesions with the advanced cordal cases.

If some such scheme of classification and staging, as suggested by I.C.P.R., was accepted internationally by both surgeons and radiotherapists it would lead to a more accurate presentation of results and comparison of results would be possible; further, results of relatively small series would be of

value, as they could be summated to give numbers which could be evaluated on a sound statistical basis.

TREATMENT.

Surgery. In this series the surgical treatment has consisted of:

1. Biopsy excision.
2. Laryngofissure.
3. Laryngectomy.
4. Radical neck dissection for metastatic nodes.

It may be pointed out that endoscopic removal was performed only once, and gave over five years of freedom before recurrence. Laryngofissure was the other conservative surgical procedure. There were no hemilaryngectomies or other modifications. In no case was a radical neck dissection performed prophylactically and only one patient suffered bilateral neck dissection. The surgery was performed mainly by Drs. Lindsay, Perlman, Schuknecht and Brunschwig.

Radiation:

1. Deep X-ray.
2. Neck mould collar (1 case).

Deep X-ray therapy has been given with machines generating 200-250 KV with H.V.L. of 1.0-1.5 mm Cu. The time dose has been 5000-5500 delivered during 35-62 days and through 5x6-10 cm field.

No cases were treated by the Finzi-Harmer fenestration technique, nor by endoscopic needling, nor by interstitial deep X-ray of Lambert and Watson techniques. Recurrences were treated by laryngectomy. Treatment was carried out under the direction of Dr. Carpender and Dr. Hamann.

So far there is very little evidence that in actual fact supervoltage machines or cobalt units will replace the standard 200, 250 or 500 KV X-ray machines in the curative radiotherapy of superficial tumors such as of the larynx, or if they do, the results will be no better, as cancer lethal doses are readily achieved by the older methods. Reduced skin dosage

and reaction is the only major advantage of the newer machines.

RESULTS.

The cases under review are a series seen in the University of Chicago Clinics up to December 31, 1949. These have, therefore, been observed, if alive, for at least five years.

Not included in the series are cases which have been partially treated at other centers and those in which there has been death from other causes. Those which have failed to return for examination or we have failed to contact, have been counted as failures as it has been assumed that they have died from the malignant lesion. This latter condition does not tend to improve the five year survival rate. The series consists of 47 cases of which a summary is presented in Table III.

TABLE III.

47 CASES UNDER TREATMENT BY SURGERY - RADIATION OR COMBINED.

Classification	No.	5 Year Survival	Per Cent
Supraglottic	11	6	54.5
Glottic	36	28	77.7
Subglottic	0	0	0

The number of cases in this series is considered still too small to give an accurate evaluation of treatment particularly when broken down into various sites and stages, but some observations can be made.

Of 11 supraglottic cases, six gained five year survival or 54.5 per cent.

Of 36 glottic cases, 28 gained five year survival, 77 per cent.

There were no cases in the subglottic group.

These overall figures confirm the well established fact that the prognosis of the glottic group is much better than that of the supraglottic group.

The glottic lesions have been broken down into stages in Table IV.

TABLE IV.

GLOTTIC CANCER.

Stages	No.	5 Year Survival	Per Cent Cure
I	9	9	100
II	25	18	72
III	1	0	0
IV	1	1	100

The excellent results in Stage I glottic cancer are at once apparent.

Of nine cases in this stage nine gained five-year survival.

There were 25 cases in Stage II and of these 18 attained five year survival, *i.e.* 72 per cent.

There was only one case in each of Stages III and IV and these, therefore, will not be studied further.

It is to be noted that 70 per cent of cases in this glottic group were in Stage II.

In Table V these nine cases in Stage I have been broken down according to the treatment received.

TABLE V.

GLOTTIC — STAGE I.

Treatment	No.	5 Year Survival
Surgery	3	3
Radiation	6	6
Combined	1	1
All cases to S	4	4

One patient treated by radiation had a recurrence at the end of six and one-half years and was then treated by laryngectomy. He is alive and well at the end of 11½ years after diagnosis.

On reviewing the results of treatment of the limited or Stage I glottic lesion by surgery and radiation at 11 centers throughout the world it was noted that there was considerable

variation in the results obtained by both types of treatment.[5,9,11,12,13,14,15,16,17,18,19]

The difference in surgical results may be mainly due to selection of cases. Some surgeons may be particularly careful to do the conservative operation only on well localized unilateral lesions while others will tend to be more optimistic. Likewise with radiation there is considerable variation which may be due to differences in experiences and technique.

If we compare the average results of surgery with the average results of radiation therapy at these various centers we find that there was only 2 per cent in favor of surgery, 88 per cent against 86 per cent, gaining five year survival.

In Table VI the glottic Stage II lesions have been broken down according to the treatment received, surgery, deep X-ray, or combined therapy.

TABLE VI.

GLOTTIC — STAGE II.

Treatment	No.	5 Year Survival	Per Cent
Surgery	11	8	72
Radiation	14	7	43*
Combined	10	7	70
All cases coming to surgery	20	14	70

* Two cases subsequently required laryngectomy thereby reducing the long term survival rate to 28.5 per cent.

Of the 11 cases or 72 per cent undergoing surgery, that is to say laryngectomy, eight obtained five year survival. In this series, one patient had a localized lesion of one cord which has caused reduced movement of the cord. Endoscopic removal gave over five year freedom from recurrence. A further period of five years has been gained by radiotherapy.

If the 14 cases receiving primary radiotherapy are examined in detail, we find that there were six five-year survivals. Two of these cases subsequently required laryngectomy and are alive and well at 10 and 16 years postoperatively. While the five year survival rate was 43 per cent, the long term cure rate was 28.5 per cent in the cases receiving radiation only.

Of the nine cases later subjected to laryngectomy six gained five year survival following surgery; thus of the 14 cases receiving primary radiation no less than 10 or 71 per cent eventually gained five year survival after secondary laryngectomy. It should be pointed out that the Group II cases which were radiated were mainly a selected group in what may be called early Stage II. Cases in late Stage II were recommended for primary laryngectomy.

Of the ten cases which received one form of treatment followed by the other when the tumor failed to respond or where there was recurrence after months or years, no less than seven or 70 per cent gained five year survival after the second form of treatment.

If we examine all cases coming to surgery we find that of 20 patients, 14, that is 70 per cent, gained five year survival, which was approximately the same as that obtained in those receiving only surgery.

From this experience, again remembering that it is only a small series, in these early Stage II cases when there is doubt as to choice of treatment between radiation and laryngectomy, or when it is imperative to save the voice, although radiation does not give as good results as surgery in Stage II, it is fairly safe to give radiation first and treat by surgery later if there is a recurrence. The percentage of five year survivals after delayed laryngectomy appeared to be as good as though surgery had been undertaken in the first instance, and in addition the patients did not lose the possibility of retaining the voice; however, the responsibility of the laryngologist is greatly increased since it is up to him to discover a recurrence at the earliest possible date. For this reason patients receiving this form of treatment must report frequently and direct laryngoscopy and biopsy must be undertaken on the slightest suspicion.

The I.C.P.R. method of classification has also been used in recent reports by Lederman[9] and Von K. Schärer.[20] Treatment of glottic carcinoma. Stage II, by radiation therapy by these authors gave five year cures in 35 and 53 per cent respectively. By comparison the results obtained in our series

were 43 per cent by radiation and 70 per cent by surgery of five year cures.

Of the 11 supraglottic cases there was one in Stage I, four in Stage II, six in Stage III, and none in Stage IV.

The overall five year survival percentage was 45. It is significant that 54 per cent of cases was in Stage III (see Table VII) as compared with 70 per cent in Stage II in the glottic group.

TABLE VII.

SUPRAGLOTTIC.

	No.	5 Year Survival
Stage I	1	0
Stage II	4	1
Stage III	6	4
Stage IV	0	0

The treatment used in the supraglottic cases is shown in Table VIII.

TABLE VIII.

SUPRAGOTTIC.

	No.	5 Year Survival
Radiation	9	2
Surgery	2	2
Combined	3	2
All coming to Surgery	5	4

It is seen that of nine cases treated primarily by radiation only two gained a five year survival; however, this group includes some cases considered to be inoperable. The two cases undergoing surgery (laryngectomy) primarily both gained five year survival and of three having surgery following radiation two were five year survivals. Due to the small number of cases in this group, it is impossible to examine the results of the two different forms of treatment in the various stages.

While remembering that some of the radiation cases may have been receiving only palliative treatment the impression is gained that surgery offers more than radiation in this group

at the present day with the help of the antibiotics and the improved techniques in neck surgery.

CONCLUSIONS.

The results attained at the University of Chicago Clinics along with those reported from other centers on the treatment of carcinoma of the larynx seem to warrant several conclusions. In the Stage I glottic lesions, excellent results can be obtained by either radiation or surgery.

In cases where the quality of the voice is an important consideration radiation offers some advantage over surgery, providing that the radiation is properly controlled and that there is a careful follow-up to detect any recurrence.

In the Stage II glottic carcinoma the results of surgery were better than those gained by radiation; however, as indicated there is a small group in the borderline between Stages I and II, where radiation may be safely tried and surgery reserved for a possible recurrence.

For some time now there has been a policy at the University of Chicago Clinics that in those cases which have passed from Stage I into borderline or early Stage II, and where the choice of treatment lies between radiation and laryngectomy and no longer between radiation and laryngofissure or some other conservative surgical procedure, radiation is advised and recurrence is treated by laryngectomy. In these cases radiation has given some good results. When there has been a recurrence, laryngectomy has given almost as good five-year survival rates as though the case had been treated primarily by surgery. This policy has had the advantage of allowing a certain percentage of such patients to retain the voice.

In the case of the supraglottic tumor, the impression gained from this series is that surgery offers a better five-year survival rate than radiation.

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MASSETER MUSCLE TREMOR: AN IMPORTANT FACTOR IN MANDIBULAR JOINT DYSFUNCTION.*†‡

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A type of bilateral masseter muscle tremor occurs in a limited number of cases being studied as mandibular joint problems. Those observed were readily differentiated from paralysis agitans and from senile tremor. Absence of mask-like expression, hand motions, and other signs appeared to prove that these belong to the senile tremor class; however, there is an age variation. Unilateral masseter tremor belongs to the degenerative palsy group, which usually shows many other obvious clinical features. Organic disease of the nervous system is absent in most cases of bilateral masseter tremor. It seems to be either an isolated muscle phenomenon, or a fraction "of oral (facial, lingual and masticatory) movements, including both spasms and tremor on a functional or psychogenic basis initiated at the subconscious level"[1] (Kempinsky).

It may not be observed at once, but may be detected by palpation with each thumb firmly pinching into the anterior border of each masseter muscle, with the patient's mouth closed. Pulsation of the facial artery is noted as synchronous with the pulse. The tremor averages about three oscillations per second; it slows under pressure of the thumb, and when palpated a moment later, has resumed a rhythmic rate. It will sometimes disappear when the patient relaxes, and returns under tension or interest.

The patient with masseter tremor describes vague dull aching of the jaws, otalgia, and other reflections of pain

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CHART I. TABULATION OF CLINICAL FINDINGS IN PATIENTS WITH MASSETER MUSCLE TREMOR

Patient	General History	ENT and Neuralgia History	Neurological Survey	Treatment	Course
Case No. 1 G. P. Age 42 Female 3-25-54	Appendectomy at age 13, cholecystectomy at 23 herniorraphy at 34, benign masses removed from ovary (L) at age 39.	Treated as eye problem 1950 to 1953. Otalgia (L) and trismus since extraction of molar teeth 1 year ago.	Dr. J. O'Leary: (1) 4-15-55: tmj syndrome. (2) chronic anxiety. (3) no neurological disorder. (4) bilateral masseter tremor, 3 cycles per second	1-14-55 occlusion balanced nine months, entirely relieved, now has occipital and parietal headache (L). 5-20-55 dental overlays removed, symptoms the same. 6-10-55 left tmj injected with 25 mgm. hydrocortisone.	Relieved nine months by balanced occlusion, overlays removed, much relieved of otalgia (L) by injection of hydrocortisone. Glossodynia (L) for three days following injection.
Case No. 2 N. F. Age 52 Female 9-25-54	Third molar extraction 1950, mild hypertension.	Four changes of partial dentures in six years, each change good for a time, then teeth strike at odd angles; numbness in tongue, cheek (R), tinnitus (R), masseter tremor bilateral.	Dr. Kempinsky: (1) contractions of facial, lingual, and masticatory muscles, at times associated with sternomastoid and platysma contraction. (2) no masseter tremor noted. (3) complete neurological entirely negative, no evidence of pseudobulbar palsy. Impression: motor neurosis or oral and facial tic probably psychogenic.	Elastic jaw splint. Ability to arrest contractions when resting.	Improved by reduction of activity at work, use of elastic jaw strap and courses of B12 injections. All symptoms remain.

CHART I. TABULATION OF CLINICAL FINDINGS IN PATIENTS WITH MASSETER MUSCLE TREMOR (Cont.)

Patient	General History	ENT and Neuralgia History	Neurological Survey	Treatment	Course
Case No. 3 S. D. Age 49 Female 1-31-51	Negative for major illness.	Extractions 1950, full dentures, tremor in jaw muscles started three months later, can be controlled by chewing gum.	Dr. Kempinsky: Gross and uneven tremor of masseters, pursing of lips, tongue movement, no sucking; neurological entirely normal; no pseudobulbar palsy. Impression: masseter tremor is part of involuntary oral movements not autonomous muscle fasciculations, can arrest them by relaxing, and has deep anxiety factor.	Elastic jaw splint; therapeutic B complex by mouth.	Symptoms remain, but improved on elastic splinting, mild sedation, and high vitamin therapy.
Case No. 4 P. L. Age 61 4-1-54 Female	Gastric studies B.H. 1948, domestic trouble basis of complaints. Treated for anorexia in gene.al medicine to date. Subtotal gastrectomy for ulcers 1950 B.H.	Teeth extracted 1945, 15 sets of dentures since. Jaw problem treated in plastic surgery 1950-51, no denture ever worn with complete comfort. Ota.gia bilat. Glassodynia rt.	Masseter tremor, thrusts tongue sideward to left, preventing denture to remain in place. Psychiatric care 1949, no neurological change.	Elastic splint attempted, cannot wear it. Anti-magnetic dentures used unsuccessfully. Seaver's myotonic exercises gave most relief.	On last visit 8-26-54 attempt to splint jaw was stopped, Seaver's exercises to continue.
Case No. 5 V. P. Age 78 Female 4-22-54	Ostructive emphysema three years, bronchial asthma, intrinsic.	Removal of nasal polyps four times in five years. Otalgia increased on coughing. Edentulous, jaws tire and plates "feel too full".	Fine tremors of masseter muscles and tongue. No neurological exam. made.	Elastic splint to jaws, 2 mm. cork between molars, bilateral.	Elastic to jaws completely relieved her, prefers to wear strap almost all the time.

CHART I. TABULATION OF CLINICAL FINDINGS IN PATIENTS WITH MASSETER MUSCLE TREMOR (Cont.)

Patient	General History	ENT and Neuralgia History	Neurological Survey	Treatment	Course
Case No. 6 I. S. Age 54 Female 8-26-54	Left condyle of mandible excised for cystic disease April 1946.	Otalgia, right, ever since resection of (L) condyle. Film shows impaction of right condyle upward.	None made. Fine masseter bilateral tremor.	Elastic fixation of jaw with 4 mm. cork within left side.	Elastic splint with 4 mm. cork with (L) side for short periods, entirely relieved, continue strap.
Case No. 7 C. O'M Age 70 Female 11-18-54	Mucous colitis. Visual disturbances, rated hypochondriac.	Otalgia, bilat. six years, pain radiates to neck and face. Most pains worse left. Films show widened flat glenoid fossa, left.	Brown-Sequard phenomenon (L) meningioma sixth thoracic spine, surgery refused. 7-9-54 bilat. masseter tremor, fine.	Elastic splint with 2 mm. cork (R). refused use of strap-pressure; too painful to face.	Refused management of obvious malocclusion. Returned at intervals to ENT clinic for "headache" up to 6-21-55.
Case No. 8 W. M. Age 80 Male 8-5-54	Remarkably negative medical history.	Pulsating sensations in rt. ear two and one-half years. no dentures worn, referred to dental clinic, narrowing of tmj, and arthritis is present bilat.	No neurological done. Audiogram shows advanced VIII nerve deafness rt. Fine tremor in tip of tongue and masseter muscles.	Elastic splint to be used on new dentures, one-half hour periods each day.	Elastic restful, uses it more than prescribed to 1-21-55.
Case No. 9 W. L. Age 85 Male 8-5-54	Hypertensive vascular disease. Emphysema enucleation right eye.	Pain in right side of face five years, typical of trigeminal neuralgia, strap gives comfort. Masseter tremor fine, bilat.	Complete neurological exam. at time of trigeminal neuralgia operation showed only senile fasciculations and trigeminal neuralgia right.	Elastic strap with 2 mm. separation right. Refer to neurosurgery operations for rt. trigeminal neuralgia 2-23-55.	Improved except for acute urinary retention and sequelae.

CHART I. TABULATION OF CLINICAL FINDINGS IN PATIENTS WITH MASSETER MUSCLE TREMOR (Concl.)

Patient	General History	ENT and Neuralgia History	Neurological Survey	Treatment	Course
Case No. 10 J. B. Age 72 Female 3-25-55	Negative except for eye problems.	Nerve deafness 40-50 dcb. bilat. Otalgia increased during eating. Crepitus in tmj bilat., tender to palpation.	Dr. Kempinsky: contractions of facial muscles, mild pursing of lips, fine tremor of extended hands, protruded tongue and masseter disease of nervous system.	Elastic splint to jaws for one-half hour periods with dentures in mouth.	Entirely relieved of otalgia by elastic splinting. The strap is restful when jaws are tired.
Case No. 11 A. McC Age 56 Female 4-15-55	Cortisone treatment of arteries in 1954, quivering of face began while in hospital, gave her speech impediment. Improved on voice therapy; divorced, has serious domestic problem.	Only catching and stopping of lower jaw on speaking or biting. Speech difficulty.	Dr. Kempinsky: orofacial tic, masseter tremor, psychoneurotic disorder, conversion type.	Elastic splint to jaws with 2 mm. cork bilaterally. Psychiatric treatment, medicine D.	Chewing much improved, uses strap shortly before eating, on 5-20-1955 reported improved by psychotherapy.
Case No. 12 J. J. Age 37 Female 2-16-55	Negative.	Coagulation rt. labyrinth for vertigo 10-8-1954, Dr. Theo. Walsh. Occipital headache, cannot wear new dentures because of nervousness. Tmjs tender to palpation, masseter tremor bilateral.	Dr. R. J. Mueller: masseter tremor, no neurological change. Referred to psychiatry for depressive state.	Elastic splinting of jaws with new dentures in place.	Splinting of jaws restful to her, masseter tremor remains, still under psychiatric care.

typical of mandibular joint disease. If old dentures are worn, they are described as loose and difficult to hold in place. With adequate new dentures the patient describes excessive tiring, and one or both dentures are left out of the mouth at intervals. This applies only to cases with the status of permanent tremor. The tiring and tremor which begins with insertion of new dentures lasts a few days and subsides, is due to the "stretch" reaction. This reaction is the basis of clonus: "If a rapid stretch is forced upon a muscle, an outburst of afferent activity results in a cycle of motoneuron responses in large numbers in step with each other. A series of clonic responses gives thus a tremor effect . . . included in this are the elevators of the lower jaw."[2] Further, in the presence of masseter tremor, when occlusion is balanced with partial dentures, striking of teeth unevenly is described after apparent perfect restoration. If this is corrected, relief is obtained for a short interval, then striking begins on the opposite side.

In this series of 22 cases, 19 are female, and three are male, ages ranging from 37 to 85 years. Twelve are briefed in the chart. The remaining ten cases are either duplications or exclude one multiple sclerosis and one pseudobulbar palsy. All have, without exception, a psychogenetic overlay, as suggested by Dr. Kempinsky. It is important to separate the muscle tremor group from those with a background of supranuclear palsy. In the latter, occlusion cannot be sustained; dentures cannot even be held in the mouth; degrees of discomfort cannot be determined. These begin with speech difficulty and ejecting of the lower denture with the tongue. There is unilateral fine masseter tremor. They progress into the hopeless stages of reflex contractions of mouth and masticatory muscles; also, conversion neuroses may not be included here, because pain descriptions are unreliable.

Treatment is symptomatic, reliance being mainly placed upon elastic splinting of the lower jaw. By simple mechanical support, this seems to relieve the muscle fatigue produced by masseter tremor. Psychiatric investigation is being carried on in selected cases and may provide a solution for some of the problems. Routine palpation of the masseter muscles is essential to early observation of this condition.

SUMMARY.

We are not so much concerned here with identifying the pain effects incidental to the mandibular joint syndrome, as with directing interest to an important muscle incoordination. Masseter tremor may be obscure when not noted in connection with facial and oral movements; it may occur as an isolated phenomenon. It is not fibrillation, nor fasciculation, nor simple muscle spasm.

The distinction between phases of oral and facial palsies which also include masseter tremor is not easy to define clinically, and actually is unnecessary; however, recognition of masseter tremor, not accompanied by other facial movements, is extremely important, completely controlling the prognosis in cases being treated for any type of mandibular joint disorder.

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THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY CONVENTION CRUISE

The Virginia Society of Ophthalmology and Otolaryngology is sponsoring a convention cruise to Havana and Nassau on May 26 to June 2, 1956. Sailing from and returning to Norfolk, Virginia, the "Queen of Bermuda" will act as the hotel for the trip. Fare for seven days, $165.00 and up per person. Make reservations with United States Travel Agency, Inc., Washington, D. C.

CLINICAL EVALUATION OF PRESBYCUSIS ON THE BASIS OF DIFFERENT TESTS OF AUDITORY FUNCTION

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The otologist who deals with elderly, hard-of-hearing patients knows how severe the hearing disability of his patients can be, even when the overall amount of hearing loss is not very great. He knows, furthermore, that two individuals with the same amount of hearing loss for pure tones can differ significantly in their perception for speech.

The present investigation is a study of the results of different tests of auditory function in a group of elderly, hard-of-hearing individuals. This study is an attempt to determine, by psychophysical measurements, medical history and findings, whether or not it is possible to improve differential diagnosis of those hearing losses which at present are included under the generic term of *presbycusis*. This term does not relate to the location of the lesions in the auditory apparatus, or to the factors responsible for the deterioration of hearing. We know from studies of previous investigators, and particularly from those of Crowe, Guild and Polvogt[1], and of Fieandt and Saxen[2], that old-age alterations in the auditory organ can be caused by different forms of disease in association with different histological lesions and pathogenesis.

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PATHOLOGY.

According to Fieandt and Saxen[2], the two most common auditory lesions are senile atrophy of the spiral ganglion cells and angiosclerotic degeneration of the inner ear. Senile atrophy of the ganglion cells is generally an independent and self-contained phenomenon, where the alterations, *i.e.*, atrophy of the ganglion cells and of the nerve fibers, affect chiefly the proximal parts of the cochlea. The organ of Corti and the blood vessels are usually not included in the process.

The angiosclerotic degeneration of the inner ear, on the other hand, begins histologically as a vascular disease. When there are lesions in the epithelium of the stria vascularis, it is possible to find marked changes in the cochlear duct, apart from a degeneration of the organ of Corti. In more advanced cases, the supporting tissue is also included in the pathological process, which is characterized by sclerosis or homogenization of the connective tissue and rarefaction of the bone tissue. The fibers of the auditory nerve are not attacked first by this process, and the changes occurring therein are only secondary.

Histologically, the atrophy of the neural elements in the cochlea corresponds to the clinical picture of presbycusis. Angiosclerotic degeneration of the inner ear alone does not produce, primarily, any important functional disturbances in the acoustic apparatus. Auditory function seems to be well-preserved in fairly advanced cases. Fieandt and Saxen[2], indicate that presbycusis may also have its origin in lesions which perhaps develop in old people on an arteriosclerotic basis in the central acoustic pathways and centers. These lesions are seldom the only reason for the defect in hearing, since in most cases they are also combined with a senile atrophy of the spiral ganglion cells and of the nerve fibers. The possibility of a central basis for the auditory defect should be taken into consideration when, in addition to the clinical picture of presbycusis, there is found a considerable deterioration of hearing for lower tones.

According to these views, supported also by the observations of Crowe, Guild and Polvogt,[1] it does not appear that vascular lesions alone can be responsible for extensive damage to the organ of Corti. Very severe lesions of the stria vascularis -

were found without apparent damage to the organ of Corti. This fact has been confirmed clinically by Bunch,[3,4] who found that the average hearing loss in patients with arteriosclerosis, hypertension, chronic cardiac conditions, syphilis and malignant conditions is not greater than the hearing loss in groups of individuals of the same age in good health.

Similar conclusions were obtained by Riesco-MacClure,[5] who found that neither clinical nor histopathologic differentiation was possible between senile hypacusis, progressive hypacusis of unknown origin in young persons, and hypacusis due to degenerative atrophic neuritis in the cochlea. Riesco-MacClure believes that cochlear alterations occurring with old age are not due to vascular changes, but result from a degenerative and atrophic neuritis.

This brief review suggests that different lesions in different locations can be responsible for those hearing losses which are subsumed under our present clinical concept of presbycusis. The literature is rich in descriptions of pathologic changes in the cochlea believed to be associated with aging; but many of them, as Covell[6] states, may be due to artifacts of fixation or postmortem autolysis, and there is also the possibility that other important features in these observations on human temporal bones may have been overlooked.

It has been pointed out previously that lesions responsible for hearing loss with age may be located in different parts of the auditory apparatus; namely, in the hair cells of the organ of Corti, in the spiral ganglion cells, in the nerve fibers, and in the central pathways. It is difficult to identify the most common locations. We can say that hearing loss in old age is a specific degenerative process with which different parts of the auditory mechanism may first be associated, and that the spiral ganglion cells and the peripheral nerve fibers are probably among the first to reveal changes.

HEARING TESTS.

We were primarily concerned with the possibility of differentiating the various types and locations of the lesions that produce deafness in old age on the basis of psychophysical

measurements. In other words, could we find some audiometric criteria to help us make a differential diagnosis?

A representative sample of elderly individuals was obtained from the files of the past two years of the Hearing Clinics at Central Institute for the Deaf. These contained extensive information on about 1500 individuals who had received hearing tests and hearing-aid evaluations. The records consisted of a case history, audiogram for air and bone conduction, monaural speech-reception threshold (spondees), monaural maximum discrimination score, binaural field-threshold for sentences, measurements of the patient's performance with several hearing aids and an otolaryngological report (where this was not completely available, a subsequent examination was carried out by one of the authors—G.P.).

From these records, a group of cases was selected that could be tentatively classified as presbycusis, according to the following criteria of evaluation:

1. Otolaryngological report negative for otoscopic and rhinoscopic findings and for previous ear diseases.

2. Case history negative with respect to previous severe diseases.

3. Sixty years of age or older (the range was from 60 to 90).

4. Audiometric indication of perceptive type hearing loss.

5. Pure-tone audiogram curve not exceeding a gradually increasing loss for higher frequencies (only in 20 cases did the slope exceed 20 db per octave).

6. Onset of deafness gradual and progressive, less than 10 years before the test.

7. Bone conduction almost equal to air conduction.

8. Profile of right and left ear air conduction almost equal, and not differing by more than 15 db at any frequency. (When the difference between the two ears was more marked, we considered this an indication of some contributing factor other than presbycusis.)

9. Average hearing loss for pure tones (500-2,000 cps) between 10 and 60 db (75 per cent of the cases have a hearing loss of between 25 and 45 db).

Only 185 cases met our criteria.

We tried to retest those subjects who resided in the St. Louis area and to do additional diagnostic audiometric tests. Of 70 persons in this category, only 24 could come back for a retest. On these subjects we carried out the following tests which lasted about two hours:

1. Short structured interview, particularly concerning past medical history.

2. Otolaryngological examination for the subjects who had not had one.

3. Pure-tone audiogram for air and bone conduction, to find whether the hearing loss had increased.

4. Recruitment test (Reger technique of monaural loudness balance between different frequencies).

5. Monaural threshold for spondees and monaural maximum discrimination score for PB lists, for test-retest reliability.

APPARATUS AND MATERIAL.

Pure-tone audiograms were obtained with a Maico H-1 audiometer. A block diagram of the equipment used for speech audiometry is presented in Fig. 1. Hearing loss for speech was measured with the Harvard Psycho-Acoustic Laboratory Test No. 9[7] spondee word lists, a set of recordings of spondaic words whose intensity decreases progressively. Speech discrimination scores were obtained by presenting phonetically balanced lists,[8] each consisting of 50 monosyllabic words, at a relatively high level. Usually, speech discrimination score was measured at a level 40 db or more above the patient's threshold for speech. When the patient could not tolerate a high sound pressure level, a lower level was used. Threshold for sentences was obtained by means of Harvard Psycho-Acoustic Laboratory Test No. 12,[7] a set of recordings of short, relatively easy sentences. This test was done in the field, and another threshold for sentences was obtained when testing the performance with a hearing aid.

The equipment diagrammed in the middle of Fig. 1 was used for monaural loudness balance test. The basic unit is an

electronic two-channel switch. Each channel was fed by a different audio-oscillator, and a mixing network delivered the two tones to the same earphone. Each tone was about one second long, with a moderately rapid but clickless rise and fall. The output of Channel 1 was alternated with that from Channel 2.

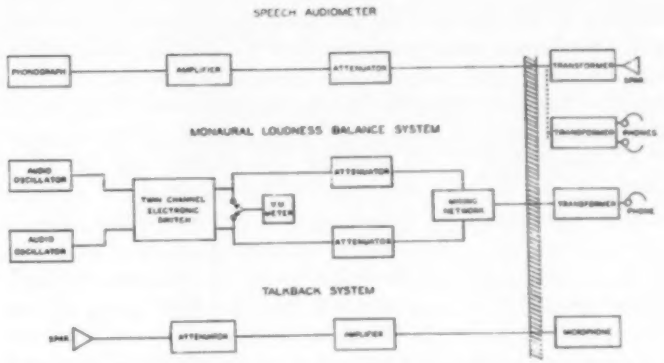

Fig. 1. Block diagram of apparatus for speech audiometry and monaural loudness-balance test.

The monaural loudness balance between different frequencies[9] was chosen as the only test for recruitment available for our particular purpose. As a matter of fact, since most of our subjects had practically the same amount of hearing loss for both ears (not exceeding 15 db difference between the two ears at any frequency), we could not use the usual Fowler[10] technique that requires not less than 25 to 30 db difference between the two ears to give consistent results. The various DL-test techniques (Lüscher[11] test and modifications, Denes and Naunton[12] and modifications) were considered but were not selected as routine tests for several reasons, chiefly on the basis of the results of Hirsh, Palva and Goodman.[13] In an experimental study based on 44 patients with a variety of types of hearing loss and 18 normal subjects, they showed that the difference limen cannot distinguish recruiting and non-recruiting cases, nor can the difference limen distinguish hard-of-hearing listeners from normal listeners,

when the DL is measured with discrete tones at sensation levels of 5, 25 and 40 db. Since negative results have also been reported by others (Lidèn and Nilsson,[14] Lund-Iversen[15]), and inasmuch as these negative results were in agreement with our own experience, we decided to utilize only the above-mentioned Reger[9] technique.

The testing procedure was usually the following: The frequencies to be matched in loudness were selected according to the pure-tone audiogram, so that the patient matched the test frequency with a reference frequency at which the hearing loss was less than 25 db. We avoided any matching of frequencies more distant than two octaves. For instance, if a subject had 15 db hearing loss at 500 cps, 25 db at 1,000 cps, 40 db at 2,000 cps, and 55 db at 4,000 cps, the balance was made by matching 500 cps and 2,000 cps. If a subject had 15 db hearing loss at 500 cps, 15 db at 1,000 cps, 25 db at 2,000 cps, and 45 at 4,000 cps, the balance was made by matching 1,000 cps and 4,000 cps. In other cases 500 cps was balanced against 1,000 cps or 1,000 cps against 2,000 cps. Frequencies of 500 and 4,000 cps were not matched because it was found that the judgment was too difficult for most subjects.

In cases where there was a hearing loss of more than 25 db, even for low frequencies, it was more difficult to demonstrate the presence of recruitment. In these cases both impaired frequencies may have had recruitment. For this reason, when the hearing loss was more than 25 db for all frequencies, we took into account only positive results indicating the presence of recruitment. Negative results we considered merely as doubtful. However, only four out of our 24 cases were in this category, and taking into consideration the threshold of pain and sharpness of response, it was possible to decide qualitatively whether or not recruitment was present.

Testing procedure was as follows: After having established the threshold for the selected frequencies, one of the tones (usually the higher) was set at 20 db above threshold; at

this time the second tone was introduced in the same ear through the circuit of the electronic switch, so that two alternating tones of one second duration each were heard by the patient. The subject was asked to tell if the "low" or the "high" tone sounded louder any time the intensity of the second tone was varied. After several measurements (usually not less than 10 or 12), balance was obtained. The same measurements were repeated at 40 db above threshold and at 60 db or more, according to the hearing loss of each patient at the tested frequencies.

RESULTS AND DISCUSSION.

All of the available data were analyzed and separated into two different groups. A first series of data includes the relations between pure-tone tests and speech tests among the total group of 185 old individuals, as taken from the record-files of the Hearing Clinics. The second series includes the relation between recruitment and discrimination score in the smaller group of 24 individuals who came back to the Clinics for the retests described above.

We emphasize that our sample of patients was "selected." It included, in fact, a group of individuals who came spontaneously to the Clinics or were referred by otologists, hearing societies, hearing aid companies, etc. In other words, this investigation was not concerned with a random sample of old people, regardless of the hearing disability of the subject, but with a selected sample of old people who came to the Clinics because they complained of being hard-of-hearing. For this reason, a comparison between our cases and the cases of some preceding World's or State Fair (New York, San Francisco, San Diego, and more recently Wisconsin) is not feasible.

Fig. 2 shows the relation between hearing loss and discrimination loss in the total group of 185 individuals. The solid line represents such a relation; on the abscissa the mean hearing loss for pure tones (500-2,000 cps) for 370 ears is indicated. On the ordinate, discrimination results are plotted in terms of discrimination "loss" instead of discrimination "score." The dotted line represents the same relation when considering the

better ear of males only, and the broken line represents the better ear of females only. There are some irregularities at the lower and at the upper ends of the scale, because of the very small number of subjects with mild (from 10 to 20 db) and severe (from 50 to 60 db) hearing loss, but in general in our population sex difference is not very significant, and the means of better ears are very close to the mean of both ears together. The 95 per cent confidence interval ranges from ± 2 per cent to ± 5.5 per cent.

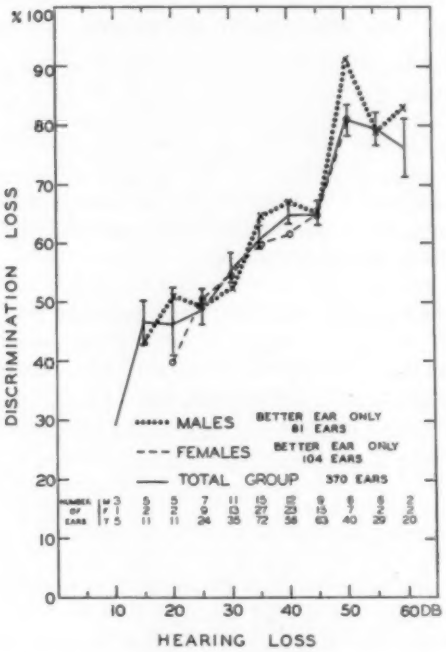

Fig. 2. Relation between discrimination loss and hearing loss in 185 old patients.

Fig. 2 shows a clear relation between hearing loss and discrimination loss. This was not expected. Discrimination losses in our old people was always very severe, even in the presence

of a mild hearing loss, and became more severe as hearing loss increased. The fact that poorer discrimination in old people is associated with a greater amount of loss has already been observed by Gaeth,[16] who, in a study on phonemic regression, found that in two groups of individuals with a discrimination "score" better than 69 per cent, the mean hearing loss was between 35 and 39 db, whereas in the group of individuals with poor discrimination (score below 68 per cent), the mean hearing loss was 51 db.

Gaeth says: "The most obvious interpretation is that phonemic regression seems to accompany more severe hearing loss. In such an event, the two deficiencies might have a common cause. On the other hand, it is possible that when a hearing loss reaches a certain degree of severity, phonemic perception is more easily disturbed. If this is true, good phonemic perception associated with severe perceptive loss may be rather exceptional." Gaeth feels, however, that an alternative interpretation is more tenable; namely, the importance of selective factors that were instrumental in bringing patients to the Clinics. In any event, the fact that poorer discrimination is associated with greater amount of hearing loss seems undoubtedly to be the case among this group of old individuals.

In order to verify this statement in other groups of individuals, we picked from our population all the individuals on whom the case history revealed the presence of some other possible contributing factors. This group was rather small, including 29 individuals: 18 with cardiovascular disorders and hypertension, eight with familial history of deafness, and three with metabolic diseases (diabetes) or chronic drug intoxications. Fig. 3 shows that the lines representing the function of the total group and of the sub-group of individuals with positive medical history have a certain amount of overlapping, and that only the groups with an average hearing loss of 35 db show a wide difference, probably due to the small number of individuals (four) in the "clinical" group with this amount of hearing loss.

The comparison between a group of old people in good health and a group of individuals with cardiovascular dis-

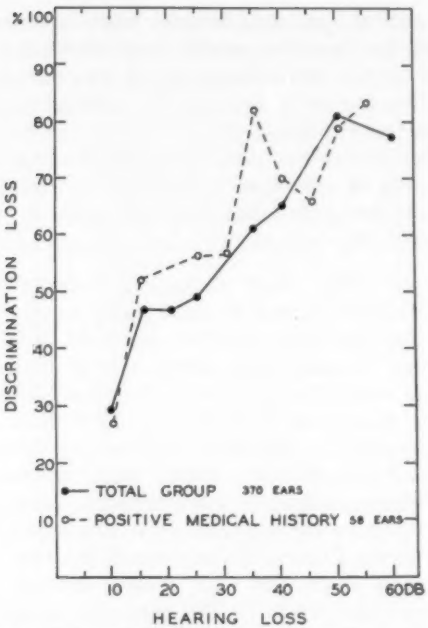

Fig. 3. Relation between discrimination and hearing loss in total group and in a sub-group showing positive medical histories.

orders, hypertension, familial history of deafness, and metabolic diseases, does not show a significant difference insofar as the relation between hearing loss and discrimination loss is concerned. Although the necessity for further more extended study is suggested, these results seem to confirm, even though considered from a different angle, Bunch's[3,4] findings, that health conditions do not influence the amount of hearing loss in people of the same age group.

Another interesting relation is revealed from the study of a second group of individuals selected from the files of the Hearing Clinics. It was our purpose to find the relation between hearing loss and discrimination loss in a group of young people, in order to ascertain whether or not the phenomenon of poorer discrimination associated with greater amount of hearing loss in a phenomenon peculiar to old age.

A group of young individuals was selected according to the following criteria: *1.* hearing loss of the perceptive type, with bone conduction almost equal to air conduction; *2.* air conduction curve of the "gradually sloping" type, showing a great similarity to presbycusis curves; *3.* overall average hearing loss for pure tones in the same range as the group of old people; *4.* not more than 40 years of age; *5.* onset of hearing loss not before 20 years of age, in order to be assured that these individuals had acquired normal development of language during childhood. Only 25 individuals met all the requirements. They had different types of hearing losses, and the clinical diagnosis according to the medical history and type of hearing loss, was possibly the following:

Heredity: three cases.

Neurologic diseases, vertigo: two cases.

Hearing loss as a result of infectious diseases: four cases.

Traumatic deafness: five cases.

Intoxication (quinine): one case.

Unknown origin (not enough information from the medical history to make a positive diagnosis): ten cases.

It can be seen that these few individuals are representative of very different types of hearing losses. There was no measure of recruitment in the records, but it is known that most hearing losses due to the above-mentioned causes are of the recruiting type. Furthermore, it is assumed that recruitment makes discrimination worse (although this is not necessarily true), but certainly not better.

From the analysis of Fig. 4 it may be noted that: *1.* Poorer discrimination associated with a greater amount of hearing loss also was present in the group of young individuals, although with less evidence; *2.* discrimination gets worse as the hearing loss goes from 10 to 30 db, but is still at the same level at 45 and 50 db hearing loss after a sudden elevation at 40 db; *3.* discrimination loss was definitely less in the group of young individuals as compared with old individuals with the same amount of hearing loss. Although the number of young individuals was smaller, the comparison between the curves

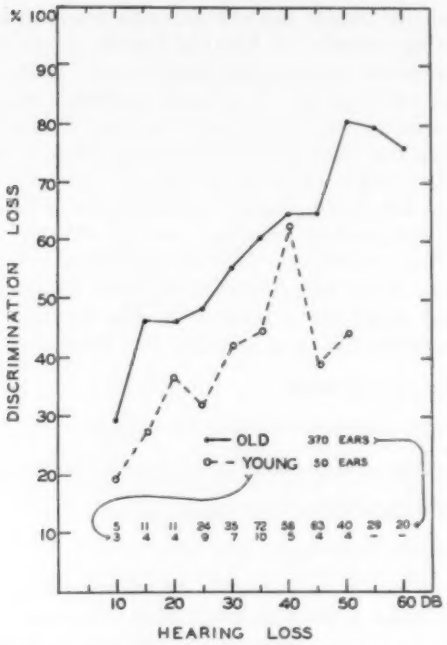

Fig. 4. Comparison of relations between discrimination loss and hearing loss in a group of old patients and in a young group with similar losses.

seems to be quite suggestive; discrimination in young people was always from nine per cent to 20 per cent better than that of the old people with the same amount of hearing loss (except at 40 db, where it is only three per cent better), and becomes almost 40 per cent better when the hearing loss is 45-50 db.

These findings seem to be a fairly good confirmation of Gaeth's[16] findings on "phonemic regression." According to this author, the phonemic-regression syndrome includes the following features: *1.* It exists under the guise of a perceptive type hearing loss; *2.* it may be more prevalent in moderate to severe hearing loss cases; *3.* it has no presently known cause; *4.* it represents a reduction in the ability to hear and to repeat common words at all supra-threshold levels; *5.* it appears to be associated with a corresponding reduction in pitch

discrimination ability; *6.* it may be complicated by low vocabulary scores.

As a comment on our findings, it is worthwhile to point out how the ability to understand speech is substantially lower than would be expected from the audiometric scores of our population. The phenomenon of phonemic regression, reported by Gaeth, substantiates this finding. Although associated with the overall amount of hearing loss even in young people, phonemic regression seems to be more clearly related to age. It is difficult to tell which pathologic phenomena are at the basis of these findings, *i.e.*, whether end-organ or central pathways are involved. We did not perform any tests of

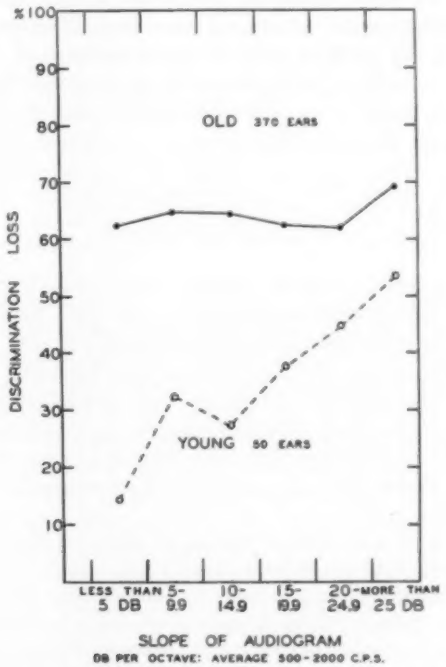

Fig. 5. Relation between discrimination loss and slope of the audiogram for group of old patients and for the young group.

intellectual functioning with our subjects, as Gaeth did (CVS tests, critical fusion frequency test, cancellation test, time reaction test). These tests, in Gaeth's population, did not show any significant deviation from the normal, even in the subjects with a very poor discrimination score. Apparently, among those subjects showing a phonemic-regression syndrome, we do not have signs of decrease of intellectual functioning to explain the poor discrimination score.

The relation between the slope of the audiogram and discrimination loss is shown on Fig. 5. On the abscissa, the slope is indicated as an average of the slope (db per octave) occurring between 500 and 1,000 cps and between 1,000 and 2,000 cps. In the group of old individuals, discrimination loss seems to be independent of the slope of the audiogram. In fact, discrimination is always very poor (ranging from 62 per cent to 64 per cent loss) in different groups with a slope less than five db per octave up to 24.9 db per octave. Only individuals with a very steep slope (more than 25 db per octave) seem to have a slightly more severe discrimination loss (69.5 per cent). In the group of young individuals, the function is different; there is a clear indication that discrimination gets worse as the slope increases.

Another relation which emerges from our data concerns the results of speech tests in relation to pure-tone tests. The hearing loss for spondees, available in 97 out of 185 individuals, was related to the average hearing loss for pure tones (500-1,000 cps and 500-1,000-2,000 cps) and the difference between speech and pure-tone hearing loss for each subject was calculated. This difference has been related to two other factors; *i.e.*, slope of the audiogram and discrimination loss.

In Fig. 6 the abscissa represents slope of the audiogram, and the ordinate represents the difference between hearing loss for spondees and for pure tones; positive numbers mean pure tones "better" than spondees, whereas negative numbers indicate pure tones "worse" than spondees. Standard deviation ranges from 5.2 db to 8.8 db. It is interesting to note that only in those patients with steep slopes of the audiogram (more

than 20 db per octave) is there good agreement between pure tone (average 500-2,000 cps) and spondees. When the audiogram is flat (less than 5 db slope per octave), or mildly sloping, the spondee hearing loss is greater than can be predicted from the average 500-2,000 cps.

It is interesting to compare our results with Carhart's[17] findings in a group of 332 individuals. He found that the range of different scores was great within every group representing different audiometric profiles (flat, gradual high tone loss, marked high tone loss, notched beyond 2,048 cps, atypical, unselected). The standard deviations for the various groups were between 9.8 and 13.8 db. Nevertheless, for a given group the mean loss in speech reception is numerically very nearly equal to the "better ear" average for 512-2,048 cps. The only exception concerns the group of individuals with marked high tone loss, where the mean hearing loss for speech is 42.4 db and the mean for pure tone average is 48.8 db. This indicates that individuals with a marked high tone loss, generally speaking, have a smaller loss for speech than can be predicted from the audiometric curve with the usual methods. Our population, as we mentioned above, deals with individuals with gradual high tone loss, but also includes some individuals with more marked high tone loss.

It is interesting to note that in our population the correlation between pure tones (average 500-2,000 cps) and speech hearing loss as a function of the slope of the audiometric curve points in the same direction as Carhart's[17] findings. The only difference is that among our population of old individuals, pure tones and spondees are almost the same when the slope of the audiogram is steeper and pure tones (av. 500-2,000) are "better" than spondees when the audiogram is flat or more gradually sloping. For this reason, we tried to correlate hearing loss for spondees with pure tones average 500-1,000 cps. only. The solid line of Fig. 6 indicates that in this way the discrepancy between spondees and pure tones becomes even greater. The function goes in the opposite direction from the case of the average 500-2,000; the steeper the slope, the greater the difference between pure tones and spondees.

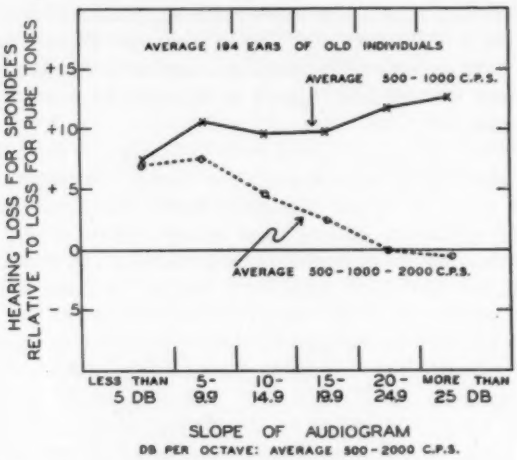

Fig. 6. Difference between hearing loss for spondees and for pure tones as a function of slope of the audiogram for a group of old individuals.

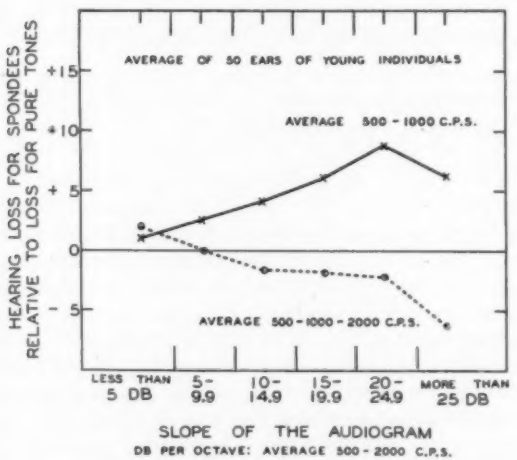

Fig. 7. Difference between hearing loss for spondees and for pure tones as a function of slope of the audiogram for a group of young individuals.

According to our data we can confirm Carhart's statement that speech reception for spondees appears to be more dependent upon sensitivity at 512 and 1,024 cps than at 2,048 cps, since the group of individuals with a steeper slope of the audiogram (*i.e.*, with a more marked loss at 2,000 cps) have a smaller hearing loss for speech than can be predicted from the average 500-2,000. The disagreement between pure tones and speech in individuals with almost flat or mildly sloping audiograms is in contrast with Carhart's results. This disagreement might be due to age difference, since the subjects in Carhart's population were unselected with respect to age.

For this reason, we examined the same relation between the slope of the audiogram and the difference between spondees and pure tones in a group of young individuals. Fig. 7 shows such a relation. Individuals with almost flat or mildly sloping audiograms (slope less than 5 db) show a better correlation between spondees and pure tones than individuals of the old group; pure tones are only from 1 to 2 db better than spondees. Furthermore, in all the individuals with a sloping audiogram (from 5-9.9 to 25 db slope per octave) hearing loss for spondees is smaller than can be predicted from the average pure tones 500-2,000 cps. On the other hand, if we correlate loss for spondees with the average loss for pure tones 500-1,000 cps, spondees show a greater loss as the slope increases.

The comparison of the relation between hearing loss for pure tones and hearing loss for spondees in young and old individuals shows that in young individuals the correlation is better. In old individuals, spondees generally show a greater loss than can be predicted from the audiogram. This finding is not surprising, since it has already been pointed out how severe discrimination loss is in old people, when tested with PB lists, as compared with discrimination loss in young people with the same amount of hearing loss.

Our results suggest that discrimination loss is significant even with easier speech material like the spondee lists. As a matter of fact, a careful analysis of the spondee scores in many of our subjects shows that many words were not understood by them even when presented at a level well above

threshold. In general, a spondee test starts from a level about 20 db above the average pure-tone threshold. At this level, normal subjects, subjects with conductive hearing loss, and even most subjects with a perceptive hearing loss understand correctly almost 100 per cent of the words. But many of our old patients did not repeat correctly 100 per cent of the words even at the level 20 db above threshold. Apparently these old people had such a severe difficulty in understanding speech that it appeared not only in the PB discrimination test, but even contributed to a greater loss for spondees. This generalization needs more careful analysis and more extensive study, but if this is true, the concept of "phonemic regression" syndrome receives still greater support and seems to be more strictly confined to the older individuals.

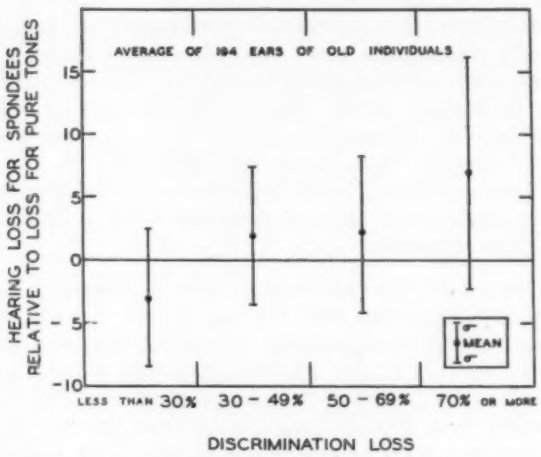

Fig. 8. Difference between hearing loss for spondees and for pure tones as a function of discrimination loss for the old group.

Fig. 8 shows the relation between discrimination loss and the difference between spondee and pure-tone loss (average 500-2,000 cps). Among four "discrimination" groups, the difference between spondee and average pure-tone loss increases as discrimination gets worse. In other words, in indi-

viduals with good discrimination (loss less than 30 per cent), spondees are 3 db "better" than pure tones; in individuals with discrimination loss from 30 per cent to 49 per cent, and from 50 per cent to 69 percent, pure tones are 2 and 3 db better than spondees, and finally in individuals with very poor discrimination (loss greater than 70 per cent), pure tones are 7 db better than spondees.

These findings further support the previous statement concerning "discrimination loss" even for spondees. When discrimination is very poor, even the threshold for spondees is shifted and there is a lack of agreement with the average hearing loss for pure tones.

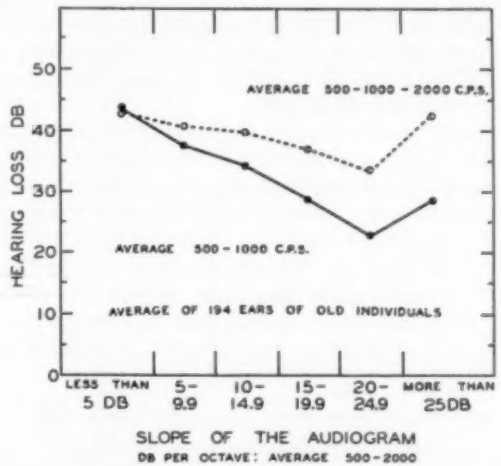

Fig. 9. Relation between averaged hearing loss for pure tones and the slope of the audiogram.

Another factor should not be underestimated when comparing the difference between average pure tones and spondee loss as a function of the slope; *i.e.*, the comparison of the amount of hearing loss for pure tones in groups of individuals with different slopes of the audiogram. Fig. 9 shows such a relation. It should be noted that, in our group, individuals with

an almost flat or mildly sloping audiogram have a more severe hearing loss than individuals with steeper slope of the audiogram. The difference is more impressive when considering hearing loss for the average pure tones 500-1.000 cps, but it is also present when we consider the average 500-2,000 cps, excluding individuals with more than 25 db slope per octave. Since we know that discrimination loss is a function of hearing loss (see Fig. 2), it is very likely that the greater difference between pure tone and spondee loss found in individuals with almost flat or mildly sloping audiograms is partially due to the larger overall amount of hearing loss shown by these individuals.

In going through our records, we noticed incidentally that it was not always possible to predict the patient's judgment as to how he liked the performance of the hearing aid. Some patients were satisfied with their aids while others were not. We decided to investigate further to see what factors were associated with this judgment.

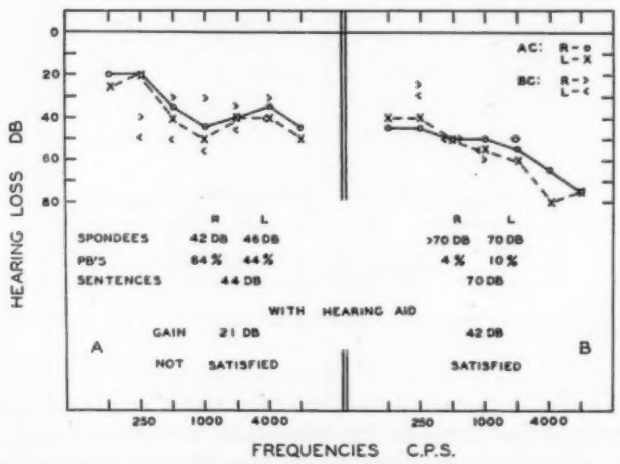

Fig. 10. Clinical records of two patients: A. has moderate loss and moderate discrimination loss and is not satisfied with a low-gain hearing aid; B. has a more severe loss with much worse discrimination and reports satisfaction with a high-gain hearing aid.

We assumed that gains obtained with hearing aids while using sentence material would be indicative of satisfaction with the performance of the hearing aid. Our results, however, do not warrant this assumption. Some patients, although they received good gains, were not satisfied with their instruments. We suspected that poor discrimination might be the influencing factor in this discrepancy. This, too, did not seem to be true with all cases, since some patients voiced dissatisfaction with their aids even though their discrimination scores were fairly good.

It appears that factors other than gains and discrimination enter into the picture. The amount of hearing loss may be the deciding factor. For example (see Fig. 10), a patient with a mild hearing loss and good discrimination *(a)* might not be satisfied with a hearing aid, while on the other hand, a person with a more severe hearing loss *(b)* might enjoy wearing an instrument even though his discrimination is very poor.

The examples cited above are extreme cases, to be sure, and many different combinations of hearing losses, discrimination scores and gains fall in between these two extremes. However, these illustrate that "scores" alone can not be used as a yardstick for predicting whether or not a patient will be satisfied with the performance of his hearing aid. Since only a small number of these patients returned for this investigation, we do not have enough information to study the relationship between "scores" and satisfaction with an instrument. This relation merits investigation on a larger sample of individuals.

Another purpose of our investigation was to ascertain whether recruitment is present in presbycusis cases, and which relations occur between recruitment and discrimination for speech. According to the data of several authors (Huizing and Reyntjes;[18] Dix, Hallpike and Hood;[19] Eby and Williams;[20] Hirsh, Palva and Goodman[13]), a low discrimination score at a high intensity level is a fairly reliable indication of recruitment. Hirsh, Palva and Goodman state that a low discrimination score will usually predict recruitment, but on the other hand, a high discrimination score does not always predict the absence of recruitment.

The relation between recruitment and discrimination loss in the 24 old patients who returned for re-test is shown in Figs. 11 and 12. Fig. 11 shows that these patients are a good sample of our population insofar as the relation between hearing loss for pure tones and discrimination loss is concerned, since this relation is approximately the same as in the total group of 185 individuals (see Fig. 2). Recruitment results have been plotted in order to attempt a quantitative rather than a qualitative judgment.

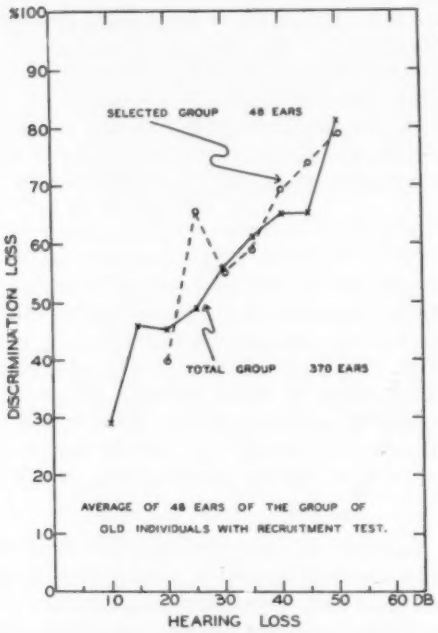

Fig. 11. Comparison of the relations between discrimination loss and hearing loss for the total group (185) and for the selected group (24) on whom further tests were made.

On the abscissa of Fig. 12, 0 db means that the same difference in db between the threshold for the two selected frequencies is still present at the maximum level (60 db or more above threshold) at which the test was performed. On the

other hand, 10 or 20 db means that at the maximum intensity level, the difference between the two selected frequencies was reduced by 10 or 20 db in respect to threshold level. We counted changes less than 5 db as no recruitment, changes between 5 and 30 db as partial recruitment, and changes more than 30 db as almost complete recruitment. According

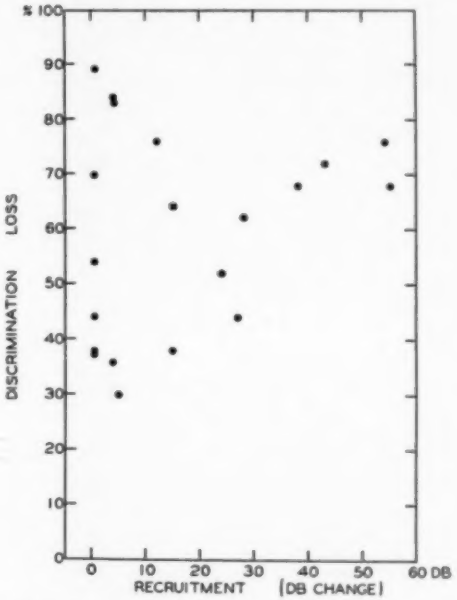

Fig. 12. Relation between discrimination loss and recruitment. Discrimination may be good or bad if recruitment is absent but is only bad if recruitment is great.

to these criteria, the following results were noted: Four subjects gave uncertain results, one of them because of his difficulty in understanding what kind of judgment was required in the test situation, three of them because their hearing losses were more than 25 db at every frequency, and recruitment may have been present at both frequencies

tested. Among these three cases, threshold of pain was lowered in one case and this might be a good argument to indicate recruitment, whereas in the other two cases, threshold of pain was not reached. These four subjects are not included in Fig. 12. From Fig. 12 it may be noted that four subjects (20 per cent) have almost complete recruitment; six subjects (30 per cent) show partial recruitment; and ten subjects (50 per cent) have no recruitment.

Discrimination is always very severe both in recruiting and non-recruiting subjects. No clear correlation is found between recruitment and discrimination loss. Nevertheless, all the subjects with almost complete recruitment have a very severe discrimination loss, whereas some of the subjects with less severe discrimination loss show no recruitment. It should be pointed out that the presence of recruitment will predict a very poor discrimination score, but on the other hand, the absence of recruitment will not predict fair or good discrimination.

COMMENTS.

One of the most important findings in our investigation seems to be that the usual relationships between different auditory tests do not hold true in presbycusis cases.

Hirsch, Palva and Goodman,[13] for instance, state that a reliable relation can exist between a low discrimination score and recruitment if the hearing loss for speech is greater than 20 db, because sometimes the hearing loss for speech frequencies is too small to affect the discrimination score, even though the loss at high frequencies is large enough to show recruitment. In our population of old individuals, this statement is no longer valid; as we can see from the previous graphs, the mean discrimination loss is severe even in the presence of a very mild hearing loss, less than 20 db.

Referring to Fig. 11, we can see that individuals with an average hearing loss of 10 db have a discrimination loss of 29.2 per cent, and individuals with 15 and 20 db hearing loss have 46.9 per cent and 46.5 per cent discrimination loss. This means that in old individuals, it is not necessary to have a hearing loss more than 20 db to affect the discrimination

score. This fact is additional support to the hypothesis of phonemic regression in old individuals. The nature of this regression is not yet clear, but many suggestions indicate that its origin is not peripheral. In fact, the absence of recruitment in most of the cases of presbycusis, substantiated also by recent papers by Harbert and Sataloff[21] and by Schuknecht,[22] suggests that discrimination score can be affected even by lesions located in parts of the auditory apparatus other than the cochlea. In other words, phonemic regression may be present in the absence of any cochlear damage. Furthermore, we know from the histologic studies mentioned above that lesions responsible for hearing loss in old people are more frequently located in the spiral ganglion cells and in the nerve fibers, rather than in the hair cells. Since recruitment is considered to be a phenomenon strictly confined to lesions of the hair cells, it seems reasonable to state that absence of recruitment in most of the cases of presbycusis provides good clinical support for histological findings.

A discrimination test alone is not of great diagnostic value in presbycusis cases if not associated with a recruitment test. Since the discrimination score is always very poor, recruitment should be the deciding factor. In the few cases that show recruitment in our sampling, we assume that lesions are more extended to the hair cells of the organ of Corti. This can be due to the process of aging itself, evolving in a different manner, or to some other reasons not fully explained by the medical history.

SUMMARY.

The present investigation has been carried out in a group of 185 selected old individuals. The relationships between different tests of auditory function gave the following results:

1. In old individuals discrimination for speech is very poor, even in the presence of a mild hearing loss. A parallel investigation in a smaller group of young individuals with the same amount and characteristics of hearing loss as the old individuals showed that young individuals have better discrimination.

2. In old individuals a clear relation exists between hearing loss and discrimination loss; discrimination becomes poorer as hearing loss becomes greater.

3. No significant relation has been found between slope of the audiogram and discrimination loss.

4. The relation between hearing loss and discrimination loss in a sub-group of individuals with positive medical history does not differ significantly from the relation in the total group of old individuals.

5. The relation between hearing loss for pure tones, hearing loss for spondees, slope of the audiogram and discrimination loss shows that individuals with a steep slope of the audiogram (more than 20 db per octave) have a smaller hearing loss for spondees than can be predicted from the audiogram (average 500-2,000 cps) with respect to individuals with almost flat or mildly sloping curves. Furthermore, in individuals with good discrimination, hearing loss for spondees is a little less than for pure tones (average 500-2,000), whereas in individuals with poor discrimination, hearing loss for spondees is definitely greater than for pure tones.

6. Since in a group of young individuals the agreement between hearing loss for pure tones and for spondees is much more precise, these results substantiate the hypothesis that in old individuals a "phonemic-regression" syndrome exists. Phonemic regression seems to have a non-peripheral basis.

7. In a smaller group of 24 old individuals in which a recruitment test was performed, 50 per cent of the subjects showed no recruitment, 30 per cent partial recruitment, 20 per cent almost complete recruitment. The absence of recruitment in most of our cases of presbycusis is in good agreement with those histological findings that indicate spiral ganglion cells and nerve fibers as the most common lesions responsible for hearing loss in presbycusis.

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BINAURAL HEARING: ANOTHER APPROACH.

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The problem of binaural summation at the threshold, or above threshold level has always raised a limited interest among otologists. On the contrary physicists, physiologists and psychologists have approached it from many angles since the time of Seebeck[13] (1846). He observed that if the whistle of a siren was led through two tubes to the two ears, it sounded weaker if one of the tubes was obstructed. Such empiric observation received further support by the work of Tarchanow[16] (1878), who, using currents produced by an induction coil connected to a telephone, noted that a subthreshold sound in one ear became audible when heard with both ears.

Urbanschitsch[17] (1893) confirmed Tarchanow's experiences by demonstrating that the induced voltage necessary to produce in a telephone a barely audible sound, needed to be twice as high in monaural hearing as in binaural hearing. Most people of that time, however, were convinced on the basis of the authority of Stumpf and Fechner, that there was no real binaural summation of intensity or of loudness but only a sensation of greater clarity and fullness of the sound; furthermore, the summation, if any, was merely physical, the intensity of two tuning forks, one in each ear, being the same as that of both forks in one ear.

An excellent review of the studies on this subject has been recently published by Hirsh,[7] and we refer the reader to this article for more detailed information. It was not until recently, as pointed out by Hirsh, that the problem of binaural summation was taken again into consideration, using more modern equipment and more reliable testing procedures.

The purpose of these new investigations was to put an end to the deadlock to which it had come as a consequence of the

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contrary evidence produced by Stumpf and Fechner on the one hand and Tarchanow and Urbantschitsch on the other.

With the exception of some studies of Fletcher and Munson[5] (1933), and of Caussé and Chavasse[3,4] (1942), related to binaural summation of loudness at intensities well above threshold, all recent work on the subject concerns almost exclusively binaural summation at threshold. The majority of the observers (Gage[6], 1932, Hughes[10], 1937, Caussé[3] and Chavasse[3,4], 1942, Shaw, Newman and Hirsh[14], 1947, Keys[11], 1947, Hirsh[7,8,9], 1948, Pollack[12], 1948), gave substantial evidence that binaural summation does exist, that it is more than physical in origin, and that at the level of the central nervous system a nearly perfect summation of the stimuli heard by the two ears takes place.

The average difference between monaural and binaural thresholds, as observed by these authors, was about 8 db both for pure tones and for speech; however Sivian and White[13] deny that there are any differences between monaural and binaural minimum audible fields which are not due to the greater sensitivity of the better ear.

This criticism has proved to be inconsistent by Caussé and Chavasse[4], and later by Hirsh, by a preliminary "equating" of the sensitivity of the two ears, so that they should be functionally equal in binaural or monaural threshold determinations. Complete agreement as to the presence, the site and the amount of binaural summation has not yet been reached.

As an otologist, I was not particularly interested in the physical issue, but some fortuitous clinical observations on patients suffering from unilateral deafness persuaded me to try a new approach to the problem of binaural hearing, the significance of which, I believe, is no less for the otologist than for the physicist.

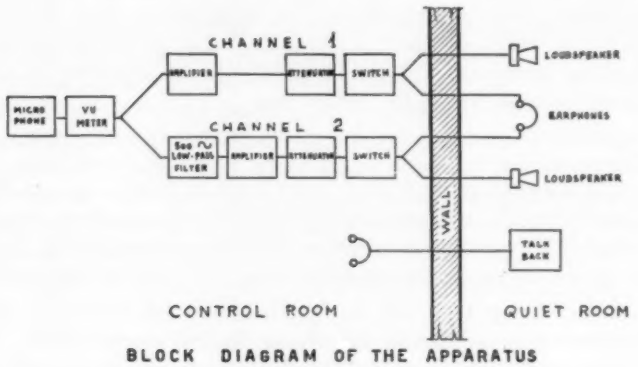
APPARATUS AND PROCEDURE.

The apparatus is shown in the block diagram of Fig. 1. It has been prepared for the present purpose by the firm, ELIT, of Milano.

Fourteen subjects were chosen for the experiment, all with normal hearing in both ears, none of them specially sophisti-

cated in auditory experiments. During the test they were isolated in a sound-proof room, and they wrote or spoke the responses according to the speech material used (logotomes or words). The test material consisted of lists of ten mixed logotomes (nonsense bisyllables of Azzi[1]) and of ten meaningful bisyllabic words (phonetically balanced lists of Bocca and Pellegrini[2]). They were spoken by a trained examiner before a microphone connected with the two channels, and the intensity of the voice was monitored by a VU meter.

Experiment 1. With the patient sitting in the room and wearing his headphones, the intensity of the voice in Channel 1 was progressively attenuated until a point was reached at which the average articulation score resulting from three determinations did not exceed 30 per cent. Subsequently



BLOCK DIAGRAM OF THE APPARATUS

Fig. 1

Channel 1 was switched off and Channel 2 was switched on. An articulation test was made on the second ear at an intensity of 45 db above threshold (threshold for obvious questions and sentences with filtered voice):

With the filter used (500 cps low-pass) articulation score never exceeded 50 per cent. At that moment both channels were switched in, and a third binaural articulation test was made. Now and again one of the two channels was switched

off, in order that the difference between monaural and binaural articulation, if any, might be further checked. The test was repeated using words instead of logotomes in another series of subjects. In half of the subjects examined the tests were entirely repeated sending the stimuli to the ear opposite to the one tested in the previous experiment.

Experiment 2. After having taken the binaural test, the subject removed his head phones and listened in succession monaurally or binaurally to two loudspeakers, one trasmitting the subthreshold normal voice, the other the suprathreshold distorted voice. Articulation was in no case higher than 50 per cent. Subsequently an articulation test was performed with the two loudspeakers acting together.

RESULTS.

The results of these experiments are illustrated in Table 1. It can be observed that both for logotomes and words, the discrimination is better when the two stimuli are delivered separately and simultaneously to the two ears. Binaural discrimination seems to be fairly equal to the arithmetic addition of the two monaural discriminations, and tends to be even higher for meaningful words.

In binaural stimulation the subject supposes he is hearing only suprathreshold stimulation, and he is unaware of the presence of the contralateral subthreshold stimulus but for the fact that the voice becomes suddenly "much clearer" when it is introduced.

When both stimuli are delivered together to one or to both ears by means of two loudspeakers, there is no effect of summation whatsoever, and the discrimination remains equal to that observed for the suprathreshold distorted stimulus alone.

The impression of the voices becoming clearer with consequent better articulation score is reached only when the intensity of the undistorted stimulus is raised at least 20 db below the intensity of the distorted one.

LOGOTOMES				
CASE	A MONAURAL DISCRIMINATION OF UNDISTORTED VOICE 5 db. BELOW THRESHOLD	B MONAURAL DISCRIMINATION FOR DISTORTED VOICE 45 db. ABOVE THRESHOLD	C MONAURAL DISCRIMINATION FOR SIMULTANEOUS DELIVERY OF A+B TO THE SAME EAR	D BINAURAL DISCRIMINATION FOR SIMULTANEOUS DELIVERY OF A+B TO THE TWO EARS SEPARATELY
1	30%	50	50	78
2	25	30	30	57
3	20	47	40	80
4	40	30	30	90
5	15	50	45	80
6	20	20	20	60
7	25	40	50	75
8	20	30	30	70
9	15	50	55	78
AVERAGE	23%	42%	40%	76%
WORDS				
CASE	A	B	C	D
1	30%	30	30	60
2	25	25	25	55
3	30	20	20	60
4	35	30	30	90
5	20	30	30	60
AVERAGE	28%	27%	27%	65%

TABLE 1

COMMENTS.

We feel that this experimental approach to the study of binaural hearing presents a number of advantages:

1. The problem of "equating" the sensitivity of the two ears is eliminated; the only problem to be solved is to choose a sufficiently low intensity of one stimulus and a sufficiently efficient filtering of the other stimulus in order that discrimination for each of them does not exceed 50 per cent, and the effect of summation may be observed in the binaural experiment.

2. The difficulty arising from any pure tone threshold determination is also eliminated by taking, as we did, as an element of judgment, not the difference in attenuation but the difference in speech discrimination score between monaural and binaural listening at levels above threshold.

3. There is evidence that the summation exists and may be considered as central in origin, as we are no longer dealing with sensitivity only, but also with discrimination. The summation effect is of nearly the same magnitude as that observed by other investigators, although using different experimental methods; *i.e.*, binaural hearing is the addition of the two monaural hearings.

4. There is no question of monaural summation, as the louder stimulus behaves exactly as a masking stimulus when delivered to the same ear together with the weaker one. To the summation phenomena observed in these experiments we would better reserve the name of binaural integration, as one ear provides power and the other provides quality.

5. The observed phenomena may help in the explanation of some unexpected good results when fitting a hearing aid on the worse ear in bilateral deafness. If the aided ear does not reach 100 per cent articulation, due to cochlear damage or to poor quality of the aid, it will be enough that in ordinary conversation subthreshold undistorted stimuli are transmitted by the other ear, to insure a higher overall discrimination by the mechanism of binaural integration, thus exceeding the results anticipated in the course of the test.

6. The test described may be of practical application to examine the efficiency of the central mechanism of hearing, even when hearing is neither normal nor equal in both ears.

SUMMARY.

An approach has been made to the study of binaural hearing by using speech as a test material. The words spoken by the examiner are delivered through two independent channels to the two ears of the subject. Channel 1 provides attenuation, Channel 2 attenuation plus 500 cps low-pass filtering. The intensity at the output of Channel 1 is adjusted each time at a level where no more than 40 per cent discrimination score is attained in repeated tests. The filter in Channel 2 does not allow more than 50 per cent discrimination score when the stimulus is presented at 45 db above threshold. If the two stimuli are presented simultaneously, one to each ear of the subject, discrimination score becomes much higher, and reaches a per cent value which is approximately equal to the addition of the two monaural discrimination scores. The experiment provides evidence of binaural summation and binaural integration.

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MIDWINTER SEMINAR IN OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Tenth Annual University of Florida Midwinter Seminar in Ophthalmology and Otolaryngology will be held at the Sans Souci Hotel, Miami Beach, the week of January 16, 1956. The lectures on Ophthalmology will be presented on January 16, 17 and 18, and those on Otolaryngology, January 19, 20 and 21. A midweek feature will be the Midwinter Convention of the Florida Society of Ophthalmology and Otolaryngology on Wednesday afternoon, January 18, to which all registrants are invited. The registrants and their wives may also attend the informal banquet at 8 p. m. Wednesday. The schedule has been changed to provide a maximum time for recreation each afternoon.

The Seminar lecturers on Ophthalmology this year are: Drs. Francis H. Adler, Philadelphia; A. Gerard DeVoe, New York; Michael J. Hogan, San Francisco; C. Wilbur Rucker, Rochester, Minn.; and A. D. Ruedmann, Detroit, Mich. Those lecturing on Otolaryngology are: Drs. Frederick A. Figi, Rochester, Minn., Lewis F. Morrison, San Francisco; Charles E. Kinney, Cleveland; John R. Lindsay, Chicago; and Bernard J. McMahon, St. Louis.

THE MODERN CONCEPT OF SURGICAL CORRECTION OF PROTRUDING EARS.*

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INTRODUCTION.

With some 30 to 40 articles in the current literature on correction of protruding ears, one might assume that no single satisfactory technique has yet been advanced. Definitely constructive trends, however, are evident in recent years. The purpose of this dissertation is to correlate, reaffirm and emphasize these and to suggest certain possible refinements.

Protruding ears constitute the most common deformity of the auricle. This fact is attested by most writers on the subject, and Becker,[5] Luckett,[22] and May[23] particularly stress it. It is also generally understood that the deformity is especially important psychologically, whether the patient be a child or an adult. The reference to "flop ears," "elephant ears," "sail ears," "donkey ears" or "Dumbo ears" is a source of constant concern to the afflicted. Children may suffer personality changes when they reach school age and are constantly ridiculed by their new-found friends and classmates. Corrective surgery may be performed at age five or six years without adverse effect on future growth. Adults often find difficulty in employment and generally in mixing with the public at large. This is one deformity which provokes laughter instead of pity, and it should be obvious that a good method of correction is important.

Otolaryngologists see more ears at close range than any other group in medical practice. They are, therefore, obliged to be equipped for correcting this most common deformity of an organ which they know from inside out, from embryo

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to old age. They should think no more of it than an orthopedist's correction of a "club foot," an ophthalmologist's correction of a ptosed eyelid or a urologist's correction of an hypospadias.

REVIEW OF LITERATURE.

The profession is greatly indebted to Becker[4] who gave a review and analysis of the literature up to 1949 along with description of the technique he used; however, there were several articles[1,2,9,11,16,23,30] with publication dates prior to that mentioned, which were not included in the review. Reference will be made to some of these. Of particular interest is a presentation by Seeley,[31] mentioned by Becker, dated 1946. This embodied a technique somewhat similar to that described by Becker and, like his, represented improvements in the then "standard" technique.

The problem of outstanding ears was recognized and written about as early as 1845 by Dieffenbach.[12] His operation advised removal of skin from the back of the ear and suturing of the ear cartilage to the mastoid periosteum. A succession of surgeons wrote further from 1881 to 1903. These were Ely,[14] Keen,[20] Monks,[25] Joseph,[19] and Morestin.[26] They removed skin from the junction of the ear with the mastoid as well as a segment of the conchal cartilage and then sutured the skin edges. This procedure must have corrected the deformity temporarily but not permanently.

Because still another type of otoplasty was suggested in 1910 by Luckett.[22] He set forth the premise, still held today, that the deformity in outstanding ears is due to the lack of an anthelix. To correct this condition, he used excision of an ellipse of cartilage and skin along the line of the new anthelix from the posterior surface of the ear. In closing, he used an everting mattress suture to roll the cartilage edges forward before closing the skin.

There followed in 1937 an article by Davis and Kitlowski[12] outlining a modification of Luckett's procedure. The approach was still to remove a large segment of skin from the mastoid and posterior surface of the ear and to remove an ellipse of

cartilage. They added an incision along the inferior crus of the anthelix and still utilized the buried everting mattress suture through the cartilage edges. Becker[5] voiced the objection that by removal of cartilage only from the inferior crus, the superior crus may appear flat and frequently allow the ear to fall forward at its upper pole.

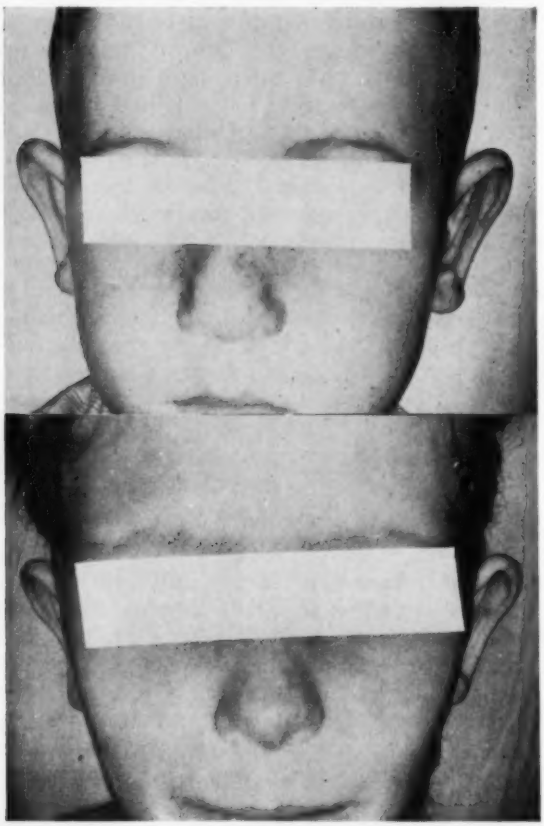

Fig. 1. Unsatisfactory results obtained by the author utilizing the technique of removal of ellipse of cartilage and skin from posterior surface of the auricle to correct protruding ears. The patients were happy but the ears lop forward from the upper pole and are "plastered" to the head at the lower pole.

New and Erich[27] (1940) added the next innovation in the form of external mattress sutures to hold the new anthelix in position while healing occurred. Their operation continued removal of an ellipse of cartilage to form the new anthelix. Segments were removed from superior and inferior crura.

With only minor differences, this technique of removal of an ellipse of cartilage and skin from the posterior surface of the ear to form a new anthelix and the application of some form of stay sutures continued for some years. It was extolled by Cox,[11] Baxter,[1] Berson,[2] Seltzer,[32] Weaver,[35] May,[23] Franklyn,[16] Rosedale,[30] Brown,[9] and Pick[28] (who removed cartilage and anterior plus posterior skin surfaces) during the 1940's. Continuing past 1950 with the same procedure advised, were Leonardo[21] (1950), Jayes[18] (1952) and in his text on Plastic Surgery (1950), Ferris Smith.[33]

Although some surgeons still use this procedure, it has definite shortcomings. Becker[5] stated the following objections: That the normal postauricular sulcus is obliterated by excess removal of skin; the superior crus may appear flat (if no cartilage is removed from it) and the upper pole of the ear may lop forward; and a sharp unnatural ridge is left on the surface of the auricle. Seeley,[31] (1946), Vidaurre[34] (1952) and Gonzales[17] (1952) found similar objections, since they advocated a departure from the then "standard" technique. The author has likewise found the technique unsatisfactory in his experience. Fig. 1 indicates how, although the ears are set back (and the patient thus made happy), there is a "dogging" from the upper pole. The auricle is plastered against the head by obliteration of the sulcus between the ear and the mastoid.

One might question: "Why bother to improve a technique which sets back the outstanding ears and thus satisfies the patient?" Cox[11] stated specifically that: "my patients repeatedly told me after operation that what they want is that the ears should be fixed at the proper angle and not 'stick-out' from the head. They do not object if the folds and contour of the pinna are not exactly the same as before." It is axiomatic that the more perfectly a restorative operation can approach normal anatomy the better. A conscientious, pres-

ent day surgeon is not satisfied with a result in which there are unnaturally sharp ridges, the pinna lops forward above, and the back of the ear is plastered to the head.

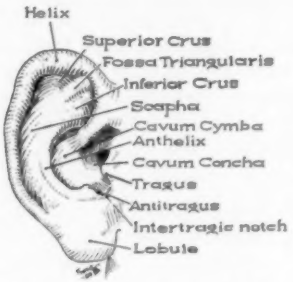

Fig. 2. External Anatomy of the Right Ear. Lateral view.

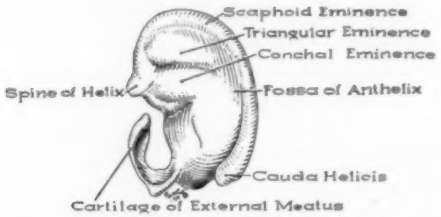

Fig. 3. Landmarks of Auricular Cartilage, Right Ear. Posterior view.

Several modifications have been advanced to get around these defections. Reference is made first to Figs. 2 and 3, which review some of the anatomical terms applicable to the external ear. One should recall that there is normally an angle of approximately 30 degrees at the upper pole between the pinna and the side of the head. The improvements suggested by Seeley,[31] Becker,[4,5,6] Vidaurre,[34] and Gonzales[17] will be considered.

Seeley[31] (1946) advised a double "Y" approach. The posterior skin was marked with methylene blue by folding the ear into the desired position. The double "Y" was marked with the long limbs joining: The fork of the upper "Y" was made by the fossa triangularis and the new anthelix crura. The

fork of the lower "Y" was made from the base of the intertragic notch anteriorly toward the cavum concha and posteriorly toward the antitragus. This double "Y" incision leaves three triangles of cartilage after skin elevation. Thin "arrowhead" segments of cartilage and skin were then trimmed so that the ear would lie back. Illustrations accompanying the article indicate good results with final contours approximating a normal ear. The author was not so fortunate, as indicated by Fig. 4.

Becker's[4,5,6] somewhat similar technique (1949-1952) was also described to set the ears back, to avoid lopping forward at the upper pole, and to avoid sharp ridges except where they appear normally. He utilized the following skin incision: starting on the posterior auricular surface near the attachment to the mastoid area, swinging laterally and then downward parallel to the helix, thence back toward the mastoid attachment. After elevation, this exposed all the posterior auricular anatomy, a point Becker justly considers important. Proposed new anthelix and roll of the helix were marked with needle punctures from the anterior surface. Incision of cartilage included the new anthelix, its inferior crus, and a peripheral incision to insure roll of the helix forward. The superior crus area was cross-cut to break the spring. A diamond-shaped wedge of cartilage was removed as needed from the anthelix and cymba. An appropriate piece of post auricular skin was removed.

Two other features he mentioned seem to be of utmost importance. The cauda helicis must be removed in most cases. A "sliver" of cartilage parallel to the upper helix rim must be removed to avoid the "dogging" forward of the upper pole. External stay sutures were applied and a small drain left in the lower end of the sutured skin incision. Fig. 4 would indicate that in the author's hands, the principles described by Seeley and Becker are not entirely satisfactory. Although the patient is happy, the ear has a plastered, unnatural appearance. Possibly the inadequacy lies in lack of proper interpretation or application of the techniques.

Vidaurre's[34] (1952) approach was again similar to Becker's up to the point of closure. Instead of using the New and

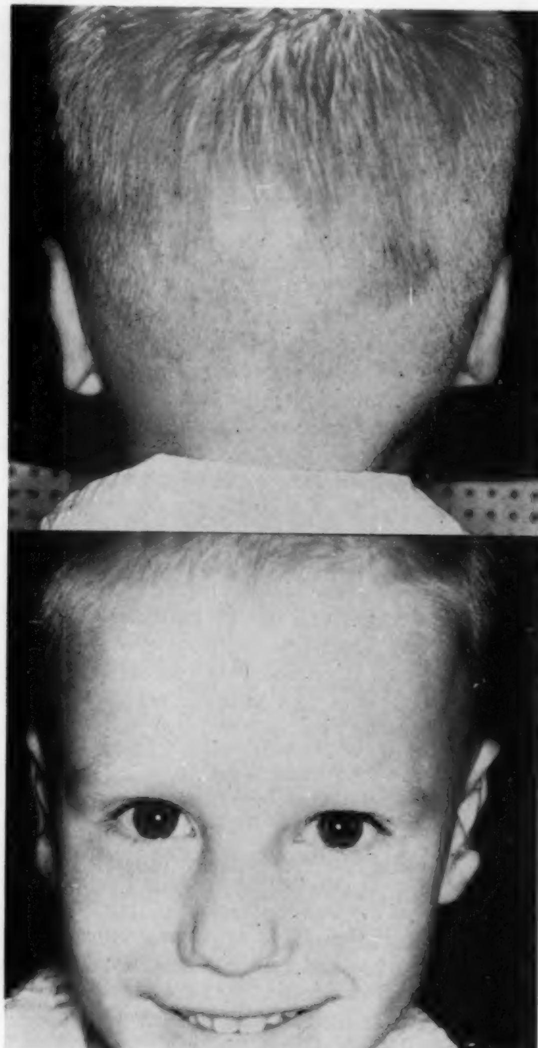

Fig. 4A.

Figs. 4—A-B. Four post-operative views of result satisfactory to patient but unsatisfactory to the author in attempt to follow principles outlined by Seeley[31] and Becker[4] to set back outstanding ears. Note "plastered" effect and sharp superior crus of anthelix. The protruding ear looks over-corrected. (Possibly the inadequacy lies in lack of proper interpretation or application of the technique).

Fig. 4B.

Erich[27] external stay sutures, he employed a type of imbrication sutures. After undermining anterior and posterior skin about the concha and removal of a cartilage section, the external segment was pulled and sutured over the medial segment in a sort of imbrication. He emphasized that the amount of cartilage removed was very important: too much makes the ear flat against the head; too little leaves a sharp anthelix.

Gonzales[17] (1952) reported an entirely different idea. On the anterior surface of the ear a five millimeter incision was made in the middle of and parallel to the anthelix. The skin was elevated through it along the new superior crus and fossa triangularis. Incision of the cartilage allowed elevation of the posterior skin. At this point small scissors were introduced and the cartilage incision extended to include the new anthelix, both the superior and the inferior crura. The small cutaneous wound was closed and filiform wire sutures utilized to hold the ear in the new position. According to pictures demonstrating the technique, there seemed to be two disadvantages: first, there appeared to be a sharp edge for the new anthelix crura; second, there was a tendency for the superior pole to lop forward (no incision made parallel to the helix as Becker described).

These in general are the modifications set forth by modern surgeons to correct the shortcomings of the Davis and Kitlowski,[12] and New and Erich[27] technique. Various small details have not been included, but the essential basis described. The chief features in common, except in the case of Gonzales, are these: incision of cartilage through the new anthelix is necessary; a segment of cartilage whether diamond-shaped or arrowhead-shaped must be removed from the cymba; some form of stay sutures are applicable whether through and through external or buried for imbrication. In addition, incision or even removal of a "sliver" parallel to the helix at its upper pole is advisable to prevent lopping forward of the upper pole. As will be seen, the author's experience would indicate these principles in general are sound.

One other question deserves discussion before proceeding to a description of detailed technique presently used by the author. That is, how to avoid a sharp, unnatural ridge at the

superior crus of the anthelix. The concensus seems to be that some form of cartilage thinning of the area as opposed to complete incision or removal of cartilage is advisable.

Pierce, Klabunde and Bergerson[29] (1947) found consistent sharp edges resulting from the removal of an ellipse of skin and cartilage from behind the ear. This was particularly evident in the area of the new superior crus fold. They stated: "Our first efforts to reproduce this fold were failures, a thinning of the cartilage from behind; but the thinning produced only another ridge:* Then came crosshatching, better, but still not satisfactory. Finally the use of eight or ten parallel incisions almost through the cartilage in the line of the desired fold gave quite an accurate representation of the normal fold."

On this point a contrary view was held by Ferris Smith[33] (1950) who still advocated removal of an ellipse of cartilage to make the new anthelix where mere incision would not suffice. He stated: "It is neither necessary to thin and bend, nor to make multiple parallel incisions ('lobster tailing') at intervals of 1 or 2 mm. along the antihelix line to produce the desired rounded edge of the new antihelix. The latter frequently suffers scar contractions and irregularities."

Becker[4,5,6,7] (1952) and Borges[8] (1953), however, agree with the concept of thinning or crosshatching the superior crus. It would seem, then, that their later agreement with Pierce et al.[29] indicates this to be a desirable refinement of technique. The author's experience would confirm this general principle.

Becker feels that cross cutting the superior crus by incisions partially through the cartilage weakens the spring to give a final smooth contour; in fact, even the cross cutting may not be necessary in some children where the cartilage is weak.

Borges gets the rounded contour of the anthelix by incising and criss-crossing it instead of removal of a segment.

It will be noted that the author prefers the uniform, better controlled thinning of the superior crus possible by using the motor driven dental burr.

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* Could this be because the thinning was not uniform, unlike one might obtain, for instance, with a dental burr?

SURGICAL TECHNIQUE.

It should be stated at the outset that the author makes no claims of originality for the technique to be described. By trial and error it has been found to work best in his hands. As noted in Fig. 1, the older technique of removal of ellipse of cartilage and posterior skin was unsatisfactory. Fig. 4 would indicate still something to be desired by adherence to Seeley's and Becker's techniques *per se* in the author's experience, granted that some lack of success might be related to difference in surgical judgment, misunderstanding or misapplication of principles laid down, etc. Suffice it to say that the technique to be described gives consistently good results when applied by the author. Every surgeon should find such a satisfactory one for himself and stick to it; so long as sound principles are applied. As will be noted, the technique utilizes certain facets of procedure from Seeley, Becker, Vidaurre, Pierce, et al., with a background of some of the earlier surgeons. It was suggested[10] that a motor driven dental burr might work well for thinning the superior crus of the anthelix to give a more natural roll. This has been tried and found useful. So far as can be ascertained at present, no one has reported experience with this minor modification to date.

Details of the modern concept of surgical correction of outstanding ears are as follows. Pre-operative pictures are made in all cases, as well as post-operative pictures. The advisability of this practice is obvious. Unlike the situation with respect to nasal plastics, pre-operative models of the ears (Brown[9]) are not considered necessary. The hair is shaved one-half inch around each ear. Surgical preparation of the skin of the ear and adjacent areas is carried out with suitable antiseptic agents, such as Phisoderm and Zepharin. The anesthesia may be either local or general depending upon the situation, but injection is made in all cases. The solution used is 1 per cent Novocaine with 5 minims of 1:1000 adrenalin and 150 units of Hyaluroindase per ounce. It has been found helpful from the standpoint of hemostasis, anesthesia and facility of dissection, to inject both anterior and posterior surfaces of the ear.

The posterior skin incision is made in the form of a "U" with the base of the resulting flap medialward at the mastoid attachment and the limbs of the "U" roughly parallel to the free edge of the auricle. It has not been found necessary to reflect perichondrium with the skin. Hemostasis is secured, though with the above mentioned injection, bleeding is almost nil.

At this point, the ear is folded back into the new position and punctures with a hypo needle made from anterior to posterior surface. The tip is touched with methylene blue and the needle withdrawn; thus one marks the posterior raw surface and the entire thickness of the cartilage but does not tattoo the posterior skin which has already been reflected. The marking should include: the new anthelix, superior and inferior crura; the section to be removed parallel to the upper helix rim (to prevent "dogging" forward); and finally the points of forward-folding of the helix rim in cases where this is flat. The average case would look somewhat like Fig. 5, from behind.

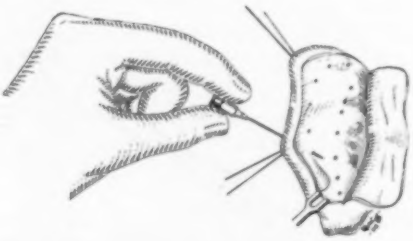

Fig. 5. Modified from Becker,[6] posterior view of left ear with skin reflected, needle punctures being made to outline areas of incision and thinning of cartilage.

The cartilage is cut through with a sharp "plastic" blade along the marked area except in the area of the superior crus of the anthelix. Finger tips of the opposite hand follow along the anterior surface to avoid cutting anterior skin. A segment of cartilage is removed from the cymba as indicated and discarded. (This cartilage has not been found suitable for use in the nasal cartilage bank). The cauda helicis is removed in all cases. In many cases part or all of the antitragus must

be removed to avoid undue protrusion of the lower pole of the ear.

In making the gentle roll of the new superior crus of the anthelix a large dental cutting burr is used for even, well controlled thinning of the area. The otologist, used to the motor driven burr for delicate dissection in mastoid and fenestration surgery, can utilize this instrument for thinning cartilage to any desired resilience at any point. Practice burr dissection on cadaver ear cartilage is deceiving. The burr thins live cartilage much more readily. An olive-shaped burr was tried but found less satisfactory than the large round cutting burr. While the opposite hand follows along the anterior skin, the cartilage is thinned from behind with the burr along the line of the new superior crus. An assistant drips cool normal saline on the burr to avoid overheating the cartilage. (This is more susceptible to the burr heat generated than is bone.) If the thinning is done along a narrow line, an anterior sharp edge will result. If it is done uniformly, wide enough to simulate the gentle curve of the normal an-

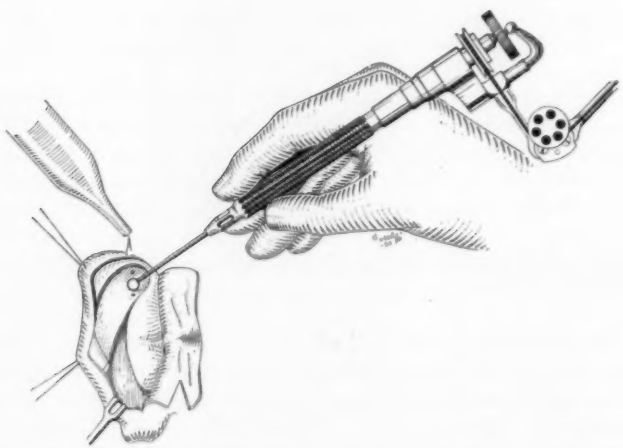

Fig. 6. Modified from Becker,[6] posterior view of dissection of left ear, indicating use of motor driven burr, cooled with saline, to thin cartilage in area of new anthelix. "Sliver" of cartilage has been removed above, parallel to helix rim and cartilage removed from area of concha. Cauda helicis and antitragus have been removed. Excess skin removal will be along line of "V" in reflected skin flap at right.

thelix, a satisfactory result will be obtained. No case to date has "suffered scar contractions and irregularities" for which Ferris Smith[23] denounced the principle of thinning this area (see Fig. 6).

Following these steps, redundant skin is trimmed away before closing. A word of caution: too generous removal of skin may imperil blood supply for healing by overly taut closure; also removal of too much skin tends to "plaster" the ear against the head when proper cartilage work has been done. True, undermining of mastoid skin could be utilized, but this should never be required by removal of too much skin.

The final step in the operation is the suturing. If the lower pole of the ear still tends to lop forward and/or upward after removal of the cauda helicis and the antitragus, a buried suture may be utilized to pull it downward and backward. It has been found expedient to insert the through and through external mattress sutures at this point before closure. The method of New and Erich[27] whereby the sutures are tied over a cotton roll is utilized; however, it is simpler to use a small roll of one-half inch "New-Gauze" plain, since it does not fray or "bunch up." Care should be exercised in tightening these mattress sutures to just the right point. If the above technique has been employed, the ear is apt to stay put in the position in which it is tied. One should be sure the 30 degree angle is present at the upper pole and that the ear is not "plastered" to the head.

A one-fourth inch flat rubber drain is inserted to prevent hematoma formation. The skin edges are approximated by fine interrupted silk sutures and the drain allowed to extend from the lower edge of the wound. Cotton pledgets soaked in mineral oil are utilized to fill in the ear contours anteriorly and posteriorly. A sterile dressing of "fluffed" 4 x 4's, two-inch roller bandage and/or "ace" elastic bandage is applied after the second ear has been operated. (While the second ear is operated, the first one is dressed loosely and the head laid downward with the ear in a "do-nut" inflated rubber ring.) The routine use of a suitable antibiotic is justified. Perichondritis may well ruin the final result, as well as increase morbidity.

Fig. 7A. Pre-operative picture.

Fig. 7A. Post-operative picture.

Figs. 7—A-B-C. Pre-operative (left hand pictures) and post-operative (right hand pictures) views show results satisfactory to patient and surgeon using technique described. The outstanding ears are set back, a 30 degree angle to the upper pole is restored and the contours approach normal.

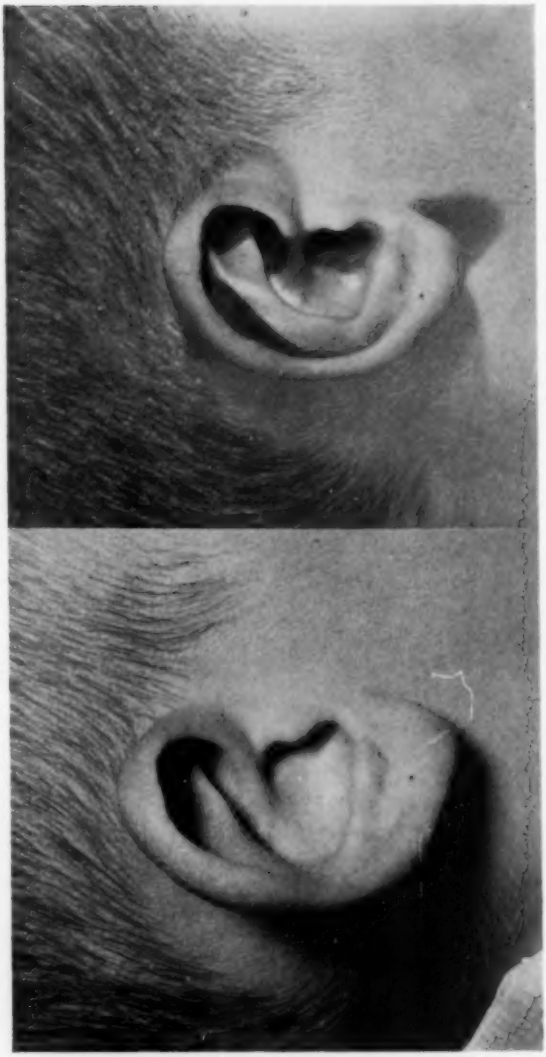

Fig. 7B. Post-operative picture.

Fig. 7B. Pre-operative picture.

Fig. 7C. Post-operative picture.

Fig. 7C. Pre-operative picture.

Fig. 8. Pre-operative picture.

Fig. 8. Post-operative picture.

Fig. 8. Pre-operative (left hand picture) and post-operative (right hand picture) views of satisfactory result obtained by the technique described. Note the gentle roll of the superior crus of the new anthelix obtained by thinning with the motor driven burr.

The dressing is not disturbed till the third post-operative day, unless the patient complains bitterly of pain, in which case the dressing is removed at once and the ears inspected for hematoma, pressure points, etc. Otherwise the drains are removed on the third day and the ears redressed. The sutures are removed on the fifth to seventh day. No pressure or elastic head band is required after the sutures are removed. If the operation has been done properly, the ears will "stay put." Figs. 7 and 8 show results of this procedure—satisfactory to the patient and surgeon. The ears are set back, a 30 degree angle to the upper pole is restored and the contours approach normal.

SUMMARY AND COMMENT.

Protruding ears constitute the most common deformity of the auricle. Since the days of 1845 when Dieffenbach[13] advised removal of skin from the back of the ear and suturing the ear cartilage to the mastoid periosteum for correction of protruding ears, numerous articles have been written. Advancement of techniques has progressed through definite steps. Next came removal of skin from the sulcus and adjacent cartilage before suturing.

Luckett[22] first recognized that the deformity in outstanding ears is due to the lack of an anthelix. His surgical correction consisted of excision of an ellipse of skin and cartilage to form a new anthelix. Everting mattress sutures (buried) were utilized. This technique (except for minor changes) with addition of external mattress sutures by New and Erich[27] was standard from 1937 until 1946.

In that year Seeley[31] described a double "Y" approach with his incisions which attempted an ear with normal contour and the proper "setting back." Becker's[4] somewhat similar technique in 1949 utilized a diamond-shaped removal of cartilage. An important addition was removal of a "sliver" of cartilage parallel to the upper helix rim to prevent lopping forward. Vidaurre's[34] solution of the problem in 1952 was similar to the latter two, except in addition he imbricated the cut cartilage medially over the conchal area to reestablish normal contour. Gonzales[17] in the same year made a complete

departure by incision of the anterior skin and cutting new anthelix folds in the cartilage. Pierce, et al.,[29] Becker,[4,5,6,7] and Borges[8] utilized thinning, or crosshatching, the superior crus of the anthelix to insure a natural roll. A sharp edge too often results from incision or removal of cartilage here.

The author finds successful results (a set back ear without sharp edges or helix lopping forward, and a natural appearing contour) by combining some operative features of Seeley,[31] Becker,[4] Vidaurre,[34] and Converse.[10] To insure uniform thinning of the superior crus of the anthelix a motor-driven burr is employed.

CONCLUSION.

Otolaryngologists, who see more ears at close range than any other group in medical practice, should be equipped to correct the most common external ear deformity—the protruding ear. The modern concept of surgical correction embodies more than removal of an ellipse of cartilage and employment of stay sutures. It establishes an ear set back, with a normal contour; no unnaturally sharp ridges and no lopping forward of the upper helix rim; it includes several incisions of cartilage, removal of segments and a uniform thinning of the superior crus of the new anthelix with a motor-driven burr.

Acknowledgments: The author acknowledges with thanks Dr. Oscar J. Becker's permission to modify and redraw some of his figures, as indicated. To Mr. Dick Mathias of Baylor University College of Medicine, the author is grateful for the medical drawings. Two of the photographs (see Fig. 8) were reproduced from print made by the Veterans Administration Hospital, Houston, Texas, Department of Medical Photography.

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SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Arrangements have been completed for the joint meeting of the North Carolina Society of Eye, Ear, Nose, and Throat, and the South Carolina Society of Ophthalmology and Otolaryngology September 17. 18, 19, 1956. Headquarters will be the George Vanderbilt Hotel, Asheville, North Carolina.

An unusually attractive program has been arranged, and a large attendance is anticipated.

Asheville, North Carolina, is in the mountains of Western North Carolina, and is a particularly beautiful spot in this season of the year.

For further information write Roderick Macdonald, M. D., Sec. and Treas., 330 East Main Street, Rock Hill, S. C.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PROGRAM 1956

Tuesday, January 3, 1956

5:30 P.M.—*Clinical Conferences

6:30 P.M.—Dinner, Parkland Hospital Dining Room

7:30 P.M.—Program:

Parkland Hospital, Rooms 101-102

Fixation of Facial Fractures Using the Kirschner Wire

Dr. Herbert H. Harris, Houston, Texas

8:00 P.M.—Technique of Cataract Extraction

Dr. Tullos O. Coston, Oklahoma City, Okla.

*Clinical Conferences at 5:30 P.M., Parkland Hospital, E.N.T. Room 103—Eye Room 104.

UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE.

The next Laryngology and Bronchoesophagology Course to be given by the University of Illinois, College of Medicine, is scheduled for the period March 5 through March 17, 1956. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois, College of Medicine, 1853 W. Polk Street, Chicago 12, Illinois.

NATIONAL SOCIETY MEETINGS.

Schedule of Meetings for 1956:

American Laryngological, Rhinological, and Otological Society Inc.:

Eastern Section, to be held at the Statler Hotel, Boston, Mass., January 13.

Middle Section, to be held at the Netherlands Plaza, Cincinnati, Ohio, January 16.

Western Section, to be held at the County Medical Society Bldg., San Francisco, Calif., January 21.

Southern Section, to be held at the Shamrock, Houston, Texas, January 27-28.

◆

American Board of Otolaryngology, to be held at the Sheraton-Mt. Royal, Montreal, Canada, May 6-11.

American Otological Society, Inc., to be held at the Seigniory Club, Montreal, Canada, May 11-12.

American Laryngological Association to be held at the Seigniory Club, Montreal, Canada, May 13-14.

American Broncho-Esophagological Association, to be held at the Sheraton-Mt. Royal, Montreal, Canada, May 15-16, (afternoons).

The American Laryngological, Rhinological and Otological Society, Inc., will hold its Annual Meeting at the Sheraton-Mt. Royal, Montreal, Canada, May 15-16-17 (mornings only).

Please make early plans to attend the 1956 Spring Meetings in Canada. Both the Seigniory Club and Montreal present most attractive features for you and your family. More information about the places will be given later.

Reservations at the Sheraton-Mt. Royal Hotel should be made early by addressing the Reservation Supervisor, 1455 Peel Street, Montreal, P. Q., Canada.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The Sixth International Congress of Otolaryngology will take place in Washington, D. C., from Sunday, May 5, through Friday, May 10, 1957, under the presidency of Arthur W. Proetz, M.D.

The selected subjects for the Plenary (Combined) Sessions to be held Monday, Wednesday and Friday mornings will be:

1. Chronic Suppuration of the Temporal Bone.

2. Collagen Disorders of the Respiratory Tract.

3. Papilloma of the Larynx.

Outstanding internationally recognized authorities will open the discussion of each of these subjects.

Two types of communications are invited: *1.* Contributions to the discussions of the selected subjects, limited to five minutes. *2.* Original papers, limited to 15 minutes. These should be in one of the four official languages: English, French, German, Spanish.

Motion picture films will be shown continuously except during the Plenary Sessions. There will be both scientific and technical exhibits. Those wishing to submit contributions to the program should communicate with the General Secretary.

Announcement of the Congress has been sent to all otolaryngologists whose names and addresses could be obtained. Additional details concerning registration, housing, entertainment, etc., will be sent to those who have indicated to the General Secretary that they wish further information.

The subscription for Members (physicians) is $25.00 U.S.A. This includes the privilege of attendance at all official Congress meetings except the banquet for which an additional charge will be made. Other persons accompanying Members may be registered as Associates at a fee of $10.00 U.S.A.

An interesting program of social functions, visits to points of interest in and around Washington and post-Congress tours is being arranged. The American Express Company is the official travel agent for the Congress. Their offices through-

out the world are available for travel arrangements to the Congress and for post-Congress tours.

All communications should be addressed to the General Secretary, Paul H. Holinger, M.D., 700 N. Michigan Ave., Chicago, Ill., U.S.A.

INDIANA UNIVERSITY MEDICAL CENTER.

The Department of Otolaryngology, Indiana University School of Medicine, offers its annual Anatomical and Clinical Course in Otolaryngology March 26th to April 7th, 1956.

Applicants should address The Post-Graduate Office, Indiana University Medical Center, Indianapolis 7, Indiana.

MOUNT SINAI HOSPITAL

Mount Sinai Hospital offers three post graduate courses given in affiliation with Columbia University. The course on Indirect Laryngoscopy is scheduled for Feb. 13-14, 1956; a course on Voice and Speech Therapy will be given Feb. 15-16, 1956 and the course in Audiology Feb. 17-18, 1956.

These courses may be taken separately or as one combined course.

For further information address The Registrar for Post Graduate Medical Instruction, Mount Sinai Hospital, Fifth Avenue and One-Hundredth Street, New York (29), N. Y.

A special course in Endaural Otologic Surgery will be given by Northwestern University February 28 through March 26, 1956. Course is limited to eight otolaryngologists. Instruction to include lectures, cadaver dissection, etc. For further information write Dr. Geo. E. Shambaugh, Jr., Dept. of Otolaryn., Northwestern Medical School, 303 East Chicago Ave., Chicago (11), Ill.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

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Vice-President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Secretary-Treasurer: Dr. Lawrence R. Boies, 90 S. Ninth St., Minneapolis 2, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Seigniory Club, Montreal, Canada, May 11-12, 1956.

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Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

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President-Elect: Dr. Percy Ireland, Toronto, Canada.
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Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Annual Meeting: Palmer House, Chicago, Illinois, October, 1956.

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Meeting: Palmer House, Chicago, Ill., October, 1956.

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General Secretary: Dr. C. E. Muñoz MacCormick, P. O. Box 9111, Santurce 29, Puerto Rico.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: April 8-12, 1956, San Juan, Puerto Rico.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.

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Place: Army and Navy Club, Washington, D. C.

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Secretary of Section on Otolaryngology: Ross A. Goodcell, M.D.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles, California.
Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section.

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